South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

Cape Town, 15 August 1959 Volume 33 No. 33 Deel 33 Kaapstad, 15 Augustus 1959

General Practice Series

THE PUERPERIUM AND ITS COMPLICATIONS

L. J. ABRAMOWITZ, M.B., CH.B. (CAPE TOWN), M.R.C.O.G.,

Department of Obstetrics and Gynaecology, University of Cape Town and Cape Provincial Administration

The puerperium is defined as the period which extends from the end of the third stage of labour until the complete return of the genitals to the non-pregnant state. Its length is from 6-8 weeks. However, there is never an absolute return to the exact pre-pregnant state, for there are permanent changes in the uterus, cervix and perineum. In common usage the puerperium refers to that period of readjustment and rest after the birth of the baby which extends over the first 2-3 weeks.

THE PHYSIOPATHOLOGY OF THE PUERPERIUM

Following normal uncomplicated labour, the general condition of the mother is good. (After complicated labour there may be varying degrees of shock, anaemia and fatigue, depending on the type of complication.) The patient is delighted with the arrival of her baby; the trials of the late first and second stage have been left behind, and there is visible delight on her countenance. A short period of apyrexial shivering often occurs. In many cases, as the days go by, emotional instability follows the mental exhilaration of childbirth. The 'maternity blues' is a well recognized entity. Patients may worry excessively about the baby. Insomnia may develop, which requires prompt attention, for the mental state may progress to one of puerperal insanity. Should the depression not respond to treatment, and become intractable, breast feeding should be discontinued.

After delivery the temperature and pulse are normal. There may be a slight rise of temperature in the first 24 hours. A further rise may occur with engorgement of the breasts, but this generally settles in 24-36 hours. Apart from this a raised temperature must be considered abnormal.

Changes in the Blood

The physiological anaemia of pregnancy is corrected and a stable haemoglobin level is reached by the 5th-7th day. In the under-privileged classes anaemia is common. This should be treated in the antenatal period, and also by prescribing a liberal iron intake in the puerperium. In severe grades of anaemia blood transfusion will be necessary. In the moderate grades imferon may suffice, but in the presence of sepsis it is wiser to enhance the patient's resistence by blood transfusion. The latter carries a small risk *per se*, and it should not be given casually without a definite indication.

The leucocyte count, which may rise to 20,000 per c.mm. during labour, returns to normal within a few days.

Gastro-intestinal Tract

After prolonged labour there may be some distension. Constipation is common in the puerperium and during lactation. In the early days it is probably due to birth trauma, and therafter it may be caused by loss of fluids by the skin, the urinary system and the breasts. Mild purgatives and glycerine suppositories produce satisfactory results in the vast majority of patients. In extreme cases enemata are necessary.

Urinary Tract

Micturition is often difficult and painful after delivery. This may result in incomplete emptying of the bladder, and a favourable environment for infection thus arises. Repeated catheterization during labour and in the puerperium also favours infection and, as always, strict aseptic precautions must be observed. If there is doubt about the complete emptying of the bladder the amount of residual urine should be assessed. If it is more than 2 oz. a self-retaining catheter should be inserted and left *in situ* until the amount of residual urine is reduced to 2 oz. or less. Urinary infection was responsible for 18 of 202 cases of puerperal morbidity which occurred in the maternity hospitals under the aegis of the University of Cape Town in 1956. This low incidence is no doubt due to a large extent to prophylactic chemotherapy. The treatment of urinary infections has been greatly simplified by antibiotics, and sensitivity tests should be performed in cases which do not respond to simple measures such as sulphonamide therapy. Stress incontinence should be treated by perineal exercises. It is often of a temporary nature and disappears with involution. In cases in which vaginal delivery has been very difficult, or where it is feared that the blood supply to the bladder may have been impaired by prolonged pressure of the foetal head, it is wise to insert an indwelling catheter for 5-7 days to prevent possible fistulae.

Genital Tract

The changes, which during pregnancy were progressive, are now retrogressive and are known as involution. Immediately after delivery the level of the fundus of the uterus is slightly below the umbilicus. It is a globular organ and it can be moved freely in the abdomen because of relaxation of the ligaments. On the first day the uterus rises slightly owing to filling of the rectum and bladder. Thereafter the level of the fundus falls so that by the 12th day it is no longer palpable per abdomen. Subinvolution occurs if placental products are retained, and in the presence of infection. The placental site decreases markedly with contraction of the uterus after delivery. Its nodular, irregular surface is virtually an open wound and may easily become infected. The tissues at the placental site exfoliate and the

surface is gradually covered by endometrium from the surrounding basal layer.

The *lochia*, which contains red blood corpuscles, leucocytes, fibrin, degenerate decidual cells and after a few days mixed organisms, are red for the first 3-4 days, and then become paler and later yellowish. Not infrequently there is a recurrence of red lochia in the first few weeks. With decomposition the lochia have a characteristic odour, and in cases of infection may become offensive.

The *cervix* is frequently lacerated during labour, and it can be seen or felt hanging loosely in the vagina after delivery. For the next few days it will admit 2 fingers and it becomes less congested. The squamous epithelium may not grow completely over the external os and frequently a cervical erosion may be found at the post-natal examination. The vagina gradually regains its tone and rugae reappear in about 3 weeks. The perineum may be lacerated and oedematous after delivery. The original tone of the perineal muscles is rarely regained completely, and there is some degree of laxity of the tissues, which may be improved with post-natal exercises.

The major complications in the genital tract in the puerperium are haemorrhage and infection.

Postpartum haemorrhage following delivery of the placenta is generally due to an atonic uterus with or without placental remnants. It may also be due to lacerations of the uterus, cervix or vagina, and rarely to afibrinogenaemia following accidental haemorrhage. The placenta must be inspected in every case to ensure that it is complete and that the possibility of placenta succenturiata does not exist. The bladder must be emptied by catheterization because uterine contraction is much improved in the presence of an empty bladder. Oxytocic drugs such as ergometrine, 0·5 mg. intravenously and pitocin, 2·5 units intramuscularly or 2·5 units given intravenously in a vacolitre of glucose water over a period, are invaluable. The uterus must be 'rubbed up' and constantly palpated to ensure that it does not relax. In the vast majority of cases the above treatment, with intravenous infusion or blood replacement, will suffice. Should they fail, or should there be evidence that the placenta is not complete, the patient should be anaesthetized and manual exploration of the uterus should be carried out, for there may be placental remains or the uterus may, in fact, be ruptured. The cervix and vagina must be inspected under a good light for possible sources of the bleeding. Bimanual compression may be of temporary value. Should the bleeding continue in the absence of any obvious cause, an intrauterine plug (6 feet of 6-inch gauze) should be inserted and left *in situ* for 48 hours under prophylactic chemotherapy. The blood-clotting time should be noted. If one is dealing with a case of afibrinogenaemia the treatment is to administer fibrinogen or fresh blood. In very rare cases the bleeding may be so intractable as to make hysterectomy a life-saving procedure.

Secondary haemorrhage (i.e. after 24 hours) is not common. Mild cases may be treated by oxytocic drugs and careful observation. In cases of recurrent or moderate bleeding the uterine cavity must be explored.

Infection. Semmelweis, as one of the early fighters in the war against puerperal sepsis, would be most impressed if he could see the progress that has been made in the battle. In 1956 there were only 105 morbid cases with genital infections among 6,898 deliveries in the maternity hospitals under the aegis of the University of Cape Town. Amongst these there were no maternal deaths. Much can be done to prevent puerperal sepsis by careful aseptic technique and prophylactic chemotherapy where it is thought to be indicated. Whatever the pathology of puerperal sepsis, all forms are treated by isolation and chemotherapy, either oral, intramuscular or in severe cases by intravenous infusion. A cervical or high vaginal swab should be taken and the organism isolated so that its drug-sensitivity can be assessed. The very rare cases of gas gangrene must be treated with antiserum and large doses of penicillin (in the region of 8-10 million units per day). It should be borne in mind that even though puerperal sepsis is now a rare cause of death, it may lead to impaired fertility and chronic pelvic invalidism.

Venous Thrombosis

Among 6,898 deliveries in 1956, there were 3 cases of venous thrombosis and 1 case of pulmonary embolism. Predisposing factors are said to be anaemia, sepsis and previous phlebothrombosis. It is highly probable that early ambulation and leg exercises reduce the incidence of thrombosis. Apart from these measures the clinician should be on constant guard for the earliest signs of the condition. Sometimes the diagnosis may be difficult. Inflation of a blood-pressure cuff to 40 mm. of mercury on the thigh may produce acute pain in the phlebitic vein of the calf. With release of the pressure the pain should dramatically disappear.[1] In the established case anticoagulant therapy should be instituted. When the calf tenderness has disappeared the patient is allowed out of bed, but anticoagulant therapy should still be continued for a number of days.

Embolic phenomena very rarely follow superficial thrombophlebitis. The response to the application of heat in one form or another and chemotherapy is excellent.

Care of the Breasts, Lactation and Breast Feeding

Attention should be paid to the breasts during the antenatal period. If the nipples are flattened or retracted, 'Woolwich' nipple shields may be worn with good effect during the last few months of pregnancy.

Shortly after delivery the baby should be placed at the mother's side. If the uterus is atonic, contraction may be stimulated by putting the baby to the breast at this early stage. Provided that the mother and baby are both well, the baby should be put to the breast once or twice on the first day, 2-3 times on the second day (for not longer than 2 minutes on each side), and thereafter 3- or 4-hourly according to the weight of the baby. Prolonged feeding on the first 2 days may result in cracked nipples since only small amounts of colostrum are present. After 48-72 hours the breasts become engorged and milk is secreted. Excessive engorgement may be very painful, and it may be relieved by the administration of stilboestrol (5 mg. twice a day for 1-2 days) and analgesics, and a comfortable brassière.

A poor milk supply may be stimulated by encouragement, a large fluid intake, the administration of Lugol's iodine, and expression of the breasts after feeds.

Cracked nipples are a trying problem in the puerperium. Apart from causing mental anguish and much physical discomfort, the condition may progress to mastitis and breast abscess. A most important aspect of treatment is the diminution of trauma to the injured nipple. For 24 hours

the baby should not be put to that breast, which should be manually expressed. Applications such as gentian violet and Friar's balsam may prove helpful. A most useful aid is the application of lead nipple shields. Occasionally the condition is so severe that breast feeding must be discontinued.

Mastitis must be recognized early and treated by chemotherapy. Once an abscess has developed, breast feeding should be discontinued and the abscess incised.

Degrees of fanaticism vary greatly with regard to breast feeding. I do not agree with the maxim, 'Breast feeding at any cost'. Difficulties do arise, and if they cannot be overcome after a fair trial, there are excellent substitutes for breast milk.

SUMMARY

Many of the physiological changes of the puerperium are discussed, in relation particularly to the mental state, changes in the blood, the gastro-intestinal and urinary tracts, the genital tract, and the breasts.

Although the fluid emotional states of the puerperium remain physiological in the vast majority of cases, the early diagnosis and treatment of psychopathic states is important.

Anaemia may result in much chronic ill-health.

The common urinary complications are, as a rule, easy to treat. One should be aware of the occasional over-distended bladder, and fistulae are more easily avoided than treated.

The causes and treatment of postpartum haemorrhage in the puerperium are discussed. The treatment of puerperal infection has been greatly simplified except with resistent organisms.

Venous thrombosis, though rare, remains a serious problem. The incidence is probably decreased by early ambulation and the prevention of infection and anaemia. anticoagulant therapy is recommended in the established case.

The problems of breast feeding such as agalactia, engorgement, cracked nipples and breast abscess are discussed.

REFERENCE

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DRAGTIGHEIDSDATA VAN *ANOPHELES GAMBIAE* GILES EN MALARIA-UITWISSING IN DIE TRANSVAAL*

J. J. STEYN, B.SC., PH.D., F.R.E.S., *Annecke Instituut, Tzaneen;* C. J. H. BRINK, B.A., M.D., D.P.H., D.T.M. & HYG., *Hoofstreeksgesondheidsbeampte, Pietersburg;* H. P. BOTHA, M.B., CH.B., *Senior Distriksgeneesheer, Pietersburg;* H. M. PRETORIUS, *Senior Gesondheidsinspekteur, Nelspruit;* H. J. COMBRINK, *Senior Gesondheidsinspekteur, Nylstroom;* en A. VOSLOO, *Tegniese Assistent, Annecke Instituut, Tzaneen.*

Weens weerstand wat verskillende vektors van malaria ontwikkel het teen nuwerwetse insektemiddels in verskeie wêrelddele, word Busvine-Nash- en Fay-weerstandstoetse aanbeveel.[3] Maar omdat dit byna onmoontlik is om in die Transvaalse Laeveld op één dag genoeg wyfies van *Anopheles gambiae*, ons vektor van malaria, vir hierdie toetse te versamel, het ons 'n moontlike vereenvoudigde dragtigheidstegniek ontwerp vir die opsporing van fisiologiese weerstand van *A. gambiae* teen BHC, waarmee Bantoehutte in die Transvaal bespuit word.[2] Gedurende hierdie ondersoek is die volgende gegewens wat nuttig is vir ons malaria-uitwissingsveldtog ook bepaal.

Tegniek

Gedurende Februarie en Maart 1958 is onbespuite hutte in die Nylstroom-substreek bedags tussen 10 vm. en 4 nm. met Pyagra bespuit vir insameling van dragtigheidsdata, en gedurende April 1958 is dragtigheidsdata ingesamel in die Nelspruit-substreek in onbespuite grondgate asook in hutte wat tussen Oktober 1958 en Februarie 1959 met BHC bespuit is.

Resultate

Die gegewens wat verkry is, is in Tabel I opgesom.

Grondgate

In Tabel I is dit interessant om daarop te wys dat daar in grondgate 80 ongevoede wyfies was teenoor 179 gevoede, halfdragtige en voldragtige wyfies.

In navorsing geld die ou leerstelling: ‚As jy nie by jou feite verbygaan nie, kom jy nooit so ver as jou feite nie.' As ons nou argumentshalwe veronderstel dat *gambiae* se dragtigheid net so lank duur as dié van ons ander lokale anophelines, sou ons soos volg kon redeneer (en later 'n ander navorser kwoteer om ons verdere beredenerings te staaf):

Veronderstel 'n *gambiae*-wyfie voed om 6 nm. en maak haar eiers in 12 uur ryp en lê daarna haar eiers. As ons dragtigheidsdata tussen 10 vm. en 4 nm. insamel, verloop dragtigheid teoreties as volg: Eerste oggend 10 vm. word gate getoets en 1 ongevoede wyfie gevind. Daardie aand om 6 nm. voed sy. Die tweede oggend om 6 vm. lê sy; sodat tussen 10 vm. en 4 nm. sy dan weer ongevoed is. As sy dus in 12 uur eiers rypmaak, sal sy altyd net in ons vangste voorkom as ongevoed, en nooit as gevoed of halfdragtig of dragtig nie. Uit Tabel I blyk dus dat sy nie in 12 uur kan ovuleer nie.

* Gepubliseer met toestemming van Die Sekretaris van Gesondheid, Pretoria.

TABEL I. DRAGTIGHEIDSDATA VAN *A. GAMBIAE* EN ANDER ANOPHELINES (10 VM.—4 NM. APRIL 1958)

Rusplekke	*Aantal rusplekke*	*Wyfies*											
		Mannetjies		*Ongevoed*		*Gevoed*		*Halfdrag*		*Voldrag*		*Totaal*	
		gam.	*an.*	*gam.*	*an.*	*gam.*	*an.*	*gam.*	*an.*	*gam.*	*an.*	*gam.*	*an.*
Grondgate	151	3	51	—	80	—	112	4	49	—	14	4	255
Onbespuite hutte†	144	37	—	13	—	30	—	41	—	59	—	143	—
BHC-bespuite hutte ..	716	12	97	6	141	19	66	10	23	—	0	35	230

† Data vir Februarie en Maart 1958; *gam.* = *gambiae; an.* = ander anophelines

As ons nou veronderstel dat ovulasie 24 uur neem, redeneer ons verder dat die verhouding van ongevoedes tot gevoedes, halfdragtiges en voldragtiges, 1 : 1 is. Maar ons verhouding was werklik 80 ongevoedes teenoor 179 gevoedes, half-halfdragtiges en voldragtiges. Derhalwe blyk dat ovulasie 179/80 dae = 2·2375 dae neem. Hierdie gegewens het 'n goeie korrelasie met die bevindings van ander navorsers insake die ovulasie van *gambiae*.

Onbespuite Hutte

In onbespuite hutte was daar 13 ongevoede en 130 gevoede, halfdragtige en voldragtige *gambiae* tussen 10 vm. en 4 nm. (Tabel II). Dus neem ons aan dat *gambiae* eers op die onbespuite muur of elders in die hut rus voordat sy voed, anders sou ons nooit ongevoede wyfies gevind het nie.

As ovulasie dus 2·2375 dae neem, kan ons redeneer dat die wyfie gemiddeld $\frac{2{\cdot}2375 \times 24 \times 13}{143}$ uur = 4·8818 uur lank in die hut rus voordat sy voed.

Hierdie syfer van 4·8818 uur is interessant want Smith[1] het bepaal dat: ‚The peak of fed *gambiae* occurs about 4 hours after the peak of the unfed, suggesting that there is a delay of about 4 hours between arrival at the enclosure wall and feeding.'

Tabel II gee 'n ander opsomming:

TABEL II. VERHOUDINGS VAN WYFIES (10 VM. — 4 NM.)

Rusplek	*Ongevoed*	*Gevoed*	*Halfdragtig*	*Voldragtig*	*Totaal*
Grondgate ..	80	112	53	14	259
			179		
Onbespuite hutte..	13 *gam.*	30 *gam.*	41 *gam.*	59	143
		71 *gam.*			
BHC-bespuite hutte	6 *gam.*	19 *gam.*	10 *gam.*	0	265
	141 *an.*	66 *an.*	23 *an.*		
	147	85	33		
		118			

gam. = *gambiae*, *an.* = ander anophelines.

As ons nou aanneem dat *gambiae* in onbespuite hutte, nes in grondgate, ook 2·2375 dae neem om eiers ryp te maak, vind ons volgens Tabel II dat sy $2{\cdot}2375 \times \frac{71}{130}$ dae = 1·222 dae neem om halfdragtig te word in onbespuite hutte.

BHC-bespuite Hutte

Tabelle I en II wys dat geen enkele *gambiae* of ander anopheline in die BHC-bespuite hutte voldragtig word nie. Derhalwe dui die data vir April 1958, nes dié vir Maart 1958[2] dat daar geen fisiologiese weerstand van *gambiae* teen BHC was nie, en dat *gambiae* doodgemaak is voordat sy voldragtig kon word.

Volgens Tabel II bereken ons verder dat as BHC *gambiae* nie doodmaak nie, daar vir elke 147 *gambiae* (en ander) ongevoedes = $\frac{71}{13} \times 147 = 802{\cdot}9$ gevoede en halfdragtiges sou wees.

Ons besluit dus dat BHC *gambiae* binne $\frac{1{\cdot}222 \times 24 \times 118}{802{\cdot}9}$ uur = 4·31 uur, uitslaan.

OPSOMMING

Ons vind dat *gambiae* blykbaar in 2·2375 dae ovuleer, 4·8818 uur lank in 'n onbespuite hut sit voordat sy byt, dat sy in 'n BHC-bespuite hut in 4·31 uur uitgeslaan word (‚knocked-down'), en dat BHC-weerstand afwesig is. Ons het reeds bepaal dat BHC-hutbespuiting 6 maande lank doeltreffend bly.[2] Malaria-uitwissing deur middel van BHC-hutbespuiting is dus goed gemotiveer.

SUMMARY

It would appear that *gambiae* ovulates in 2·2375 days, sits for 4·8818 hours in an unsprayed hut before feeding, and is knocked down in a BHC-sprayed hut in 4·31 hours. It has already been determined that spraying huts with BHC remains effective for 6 months. Malaria eradication by means of spraying huts with BHC is therefore based on a sound rationale.

Graag bedank ons elkeen van die veldpersoneel wat dragtigheidsdata in die Nylstroom- en Nelspruit-substreke ingesamel het vir hierdie navorsing.

VERWYSINGS

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2. Steyn, J. J., Brink, C. J. H., Botha, H. P., Pretorius, H. M., en Combrink, H. J. (1959): S. Afr. T. Geneesk., 33, 172.
3. World Health Organization (1958): *Seminar on the Resistance of Insects to Insecticides*. Wld Hlth Org. Techn. Rep. Ser. No. 76.

42ND MEDICAL CONGRESS (M.A.S.A.), EAST LONDON, 27 SEPTEMBER–3 OCTOBER 1959
42STE MEDIESE KONGRES (M.V.S.A.), OOS-LONDEN, 27 SEPTEMBER–3 OKTOBER 1959

Distinguished Visitors to Congress

The Organizing Committee of Congress have announced that the wife of Prof. Dr. T. Antoine, of Vienna, will also attend Congress and take part in the scientific proceedings. Dr. Lore Antoine is a dermatologist and Vice-president of the International Federation of Medical Women.

Alterations to Programme

The Organizing Committee have made the following alterations to the Programme of Congress as published in the *Journal* of 25 July (**33**, 629):

Plenary Session

Wednesday a.m., 30 *September*

HEART DISEASE

(*a*) *Coronary disease*. The aetiology and diagnosis of ischaemic heart disease: Prof. G. Burch (USA).

The prophylaxis and management of coronary heart disease: Prof. J. F. Brock.

The treatment of coronary heart disease: Dr. M. M. Suzman.

(*b*) *Cardiac surgery*. Cardiac surgery: Sir Russell Brock (UK). Late results of mitral valvotomy: Mr. L. Fatti.

South African Medical Journal : Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL : VAN DIE REDAKSIE

'COMPLICATIONS' OF DIABETES

If we were asked, 'What is diabetes mellitus?', we should probably all give some sort of answer based on high blood sugar, loss of tolerance to glucose or, more vaguely, abnormalities of carbohydrate metabolism. It could, however, well be argued that, from the patients' point of view, the metabolic derangements are no longer of great importance. The loss of health and of life associated with ketosis is now very largely abolished by the use of insulin. But, lurking in the background in all diabetics, whether mild or severe, is a much more sinister bogey—the hazard of vascular disease, bringing with it blindness, gangrene, heart disease, renal failure, and death.

As a working hypothesis we may consider the retinopathy and the Kimmelstiel-Wilson nephropathy as specific diabetic lesions affecting tiny blood vessels; the peripheral vascular lesions in the diabetic which affect small arteries may also be in part specific phenomena;[1] coronary atheroma is increased in diabetes—most markedly in pre-menopausal diabetic women, in whom the incidence of ischaemic heart disease is equal to that of men of the same age; and the neuropathy most likely has a vascular basis, although this cannot be considered proved. The outstanding questions concerning these lesions are, first, what is their pathogenesis and what relation do they bear to the hyperglycaemia and other metabolic aspects of diabetes and, secondly, how can they be prevented or ameliorated?

With regard to the first question, many people are coming to believe that these lesions are not 'complications' of diabetes at all, but are integral features of the innate disease. In other words, when one inherits the genetic 'tendency' to diabetes, this automatically includes a deficiency in blood vasculature likely to lead to the conditions mentioned above. If this is so it becomes plain that 'control' of the hyperglycaemia of diabetes, however good, cannot be expected to prevent the vascular disorders. Nevertheless, the evidence is now rather convincing that good diabetic control does tend to lessen the likelihood that vascular disease will appear and to reduce its severity after it has appeared. Indeed it is really in this belief that we bother to control diabetes at all beyond maintaining well-being and preventing hypoglycaemic attacks. Impressive recent evidence has been provided by Professor Dunlop,[2] of Edinburgh, who changed over from the use of free 'diet-and-let-control-go-hang' to careful control, and found a gratifying reduction in vascular disease.

In an article published in this issue of the *Journal*, Markman, Allen and Jackson[3] analyse the findings in a sample of the patients attending the Diabetic Clinic at Groote Schuur Hospital, Cape Town. It is a pity that the sample could not be larger, but surveys of this nature are not easy to organize and are immensely time-consuming. As might be expected, the control of the diabetes was in general poorer in non-Europeans (they were almost all Cape Coloured) and there was a higher incidence of retinopathy in this group, particularly in females. The incidence of vascular disorder was probably much the same as reported in Britain and America, though perhaps lower if anything—for instance, evidence of nephropathy was very seldom found. Poor control of diabetes appeared to carry with it a significantly higher incidence of retinopathy and neuropathy, but made no difference to the amount of coronary heart disease. This finding is important; it is presumably related to the fact that retinopathy and neuropathy are specific diabetic lesions, whereas coronary atheroma in individual diabetics differs in no way from atheroma in non-diabetics. In this series the sex incidences of coronary heart disease are virtually equal, suggesting, as has been seen from larger evidence elsewhere, that there is something in diabetes that annuls the advantage of being born a woman!

It appears that, in general, there is little or no difference between the diabetes in the European and that in the Cape Coloured population. We await with great interest a similar comparison which will include the Bantu—and also the West African and the American Negro—which, as far as we are aware, has not yet appeared, despite the great need for it and the ease with which one part of it could be carried out in the United States.

1. Lundbaek, K. (1953): Acta med. scand., 145, suppl. 277.
2. Dunlop, D. M. (1954): Brit. Med. J., 2, 383.
3. Markman, P., Allen, E. A. and Jackson, W. P. U. (1959): S. Afr. Med. J., 33, 682.

EMOSIONELE DEELNAME EN DIE DOKTER

Anders as wat die geval is met die meeste ander professies, bring die spesiale soort taak van die dokter dit mee dat hy gedurig diep verwikkel raak in die emosionele en persoonlike reaksies en spanninge van die pasiënte met wie hy werk, sowel as van hul naasbestaandes en bekendes. Hierdie emosionele deelname, wat 'n onafskeidbare deel vorm van die werk van die dokter, lê 'n groot addisionele las op hom. En, in die gevalle van baie dokters is dit nie soseer die hoeveelheid en die omvang van hulle werk as sodanig wat tot vermoeienis lei nie, maar wel die eise wat aan hulle gestel word ten opsigte van die emosionele deelname aan al die innerlike leed en verwarring en ontreddering en hulpbehoewendheid waaraan hul pasiënt van tyd tot tyd blootgestel is.

Dit moet vir almal duidelik wees dat die dokter nie kan toelaat dat sy psigiese energie heeltemal verteer word deur te diepe verwikkeling met die emosionele lewe van elke pasiënt wat hy behandel nie. Want dan sal sy doeltreffendheid as mens en as dokter skade ly en sal hy tot 'n mindere mate in staat wees om in elke geval sy beste dienste te lewer. Die

dokter moet dus in staat wees om in 'n sekere mate afsydig te staan teenoor wat om hom gebeur, sonder om egter onsimpatiek te wees.

Om hierdie balans tussen gesonde afsydigheid en noodsaaklike deelname te behou, is geen geringe taak nie. Dit vereis 'n ryp en verstandige gees by die dokter en insig in dié uitspraak: ‚mens wees—dit is die groot gebod'. Hoe kan dit anders, want die dokter moet sy werk doeltreffend doen in die aansig van sulke omstandighede soos die verlies van werk en finansiële ineenstorting by sy pasiënte, gebroke gesinne, emosionele versteuring, wanaanpassing, verminking, liggaamlike siekte, en die dood.

In die verlede het elke wyse en verstandige dokter hierdie probleem op sy eie manier benader en opgelos. En, in soverre as wat hy daarin geslaag het om die delikate balans tussen meelewing en die behoud van eie kragte te bereik, is hy deur sy pasiënte beskou, nie net as 'n goeie dokter nie, maar ook as 'n groot en begenadigde mens. Met die opkoms van wat ons kan noem die moderne ‚industriële revolusie' in die medisyne, het hierdie basiese probleem egter na 'n ander vlak verskuif. Spesialisasie en hiperspesialisasie, wat aan die een kant lei tot byna foutlose, maar tog robotagtige, mediese dienste, lei aan die ander kant ook daartoe dat die persoon van die dokter al meer op die agtergrond raak.

Wat ons nou net gesê het is veral waar van verskeie gespesialiseerde vertakkinge van die medisyne, maar ook van sekere maniere waarop die algemene praktyk bedryf word. 'n Angstige, ontstelde pasiënt word soms onderwerp aan 'n hele reeks toetse en ondersoek-prosedures sonder dat daar genoegsame persoonlike kontak tussen hom en sy dokters is. Wat die oplossing van hierdie probleem is, weet ons nie. Wat ons egter wel weet, is dat dit in terme van die uiteindelike welsyn van die pasiënt—liggaamlik en geestelik—nie 'n gesonde toestand van sake is nie. Die behoefte van die mens aan 'n intieme vertroueling is nou eenmaal te diep gewortel om so summier misken te word. En die dokter van die toekoms sal op een of ander manier 'n formule moet vind wat dit vir hom moontlik sal maak om te voldoen aan die vereistes van die moderne tegniese mediese praktyk sonder om sy pasiënt as mens en vertroueling van hom te vervreem.

AN ANALYSIS OF THE RETINAL, CARDIOVASCULAR AND NEUROLOGICAL DISORDERS IN DIABETICS ATTENDING AN OUT-PATIENT CLINIC

P. MARKMAN, F.C.P. (S.A.), M.R.C.P. (LOND.); E. A. ALLEN, M.B., CH.B. (CAPE TOWN); AND W. P. U. JACKSON, M.A., M.D. (CAMB.), M.R.C.P. (LOND.), D.C.H.

Diabetic Clinic, Department of Medicine, Groote Schuur Hospital and University of Cape Town

It is the purpose of this paper to report a preliminary analysis of the findings derived from a study of diabetic patients in Cape Town. For about 18 months a number of patients seen at the Groote Schuur Hospital Diabetic Out-patient Department have been asked to attend a special clinic held each week. At these sessions relevant historical data are recorded and a clinical and electrocardiographic examination is carried out. At each session an ophthalmologist has attended to record the ocular findings.

As the cases for this special clinic are largely selected at random by a social worker it is fair to presume that they are representative of the diabetic patients attending the hospital as out-patients. Approximately 2,000 patients are on the lists of the Diabetic Out-patient Department at present and of these about 200 attend the clinic each week.

The retinal, cardiovascular and neurological complications* occurring in 210 consecutive patients are considered in this analysis.

The Series as a Whole

The patients under review comprise 129 Europeans and 81 non-Europeans (54 Cape Coloured, 24 Malays and 3 Bantu).† 149 are female and 61 male, reflecting the greater number of women attending the Diabetic Out-patient Department as a whole.

Their distribution in 4 age-groups is as follows:

Group A: 19 years and under, 12 patients
Group B: 20 to 39 years, 20 patients
Group C: 40 to 59 years, 83 patients
Group D: 60 years and over, 95 patients.

In 156 patients the duration of diabetes was under 10 years and in 52 it was 10 years or longer.

As an arbitrary index of the severity of the diabetes the patients are divided into 4 groups according to their insulin requirements at the time of examination.‡ Their distribution in the 4 groups is as follows:

Group 1: Diet only, 54 patients.
Group 2: Diet and up to 20 units of insulin daily, 32 patients.
Group 3: Diet and 21 to 50 units of insulin daily, 81 patients.
Group 4: Diet and 51 to 200 units of insulin daily, 43 patients.

In none of the patients was the insulin requirement larger than 200 units daily.

Obvious difficulties are encountered in attempting an assessment of diabetic control. In the present series we have divided the cases into 4 groups, showing excellent, good, fair and poor control respectively. The assessment has been based on the following criteria:

Excellent: No hypoglycaemia. Glycosuria never more than 2 plus, usually nil. Fasting blood sugar 90-140 mg.%.
Good: Hypoglycaemia extremely mild and rare. Glycosuria nil to 2 plus, rarely 3 or 4 plus. Fasting blood sugar in the region of 170 mg.%.

* The vascular lesions of diabetes are probably not true 'complications'—they are more likely an integral part of the disease itself, but nevertheless the term is in general accepted and is convenient for use in this paper.

† For overseas readers: 'European' refers to all 'White' (Caucasian) persons. 'Non-European' refers to all others. 'Cape Coloured' refers to the Cape Province half-caste mulatto. 'Malays' are a special variety of the Cape Coloured, and are Mohammedans.

‡ The insulin requirement is no true indication of the severity of the diabetes, since a juvenile, ketosis-prone diabetic may take only 20 units, and a mild, obese, relatively resistant oldster may be uncontrolled on 200 units. Nevertheless we include analyses on this basis for want of a better one.

Fair: Occasional hypoglycaemia. Glycosuria variable, often 3 or 4 plus. No ketosis.
Poor: Liable to hypoglycaemia or ketosis or both not infrequently. Glycosuria constantly 4 plus.

In 197 patients adequate data was available for an evaluation of control. This was assessed as excellent in 57, good in 70, fair in 35, and poor in 35 patients. The distribution of the control assessment separately considered for the two main racial groups can be seen in Table I.

TABLE I. THE RELATION BETWEEN RACE AND CONTROL

Control	*Europeans (White)*		*Non-Europeans (non-White)*	
	No.	*Percentage*	*No.*	*Percentage*
Excellent	45	34·9	12	17·7
Good	48	37·2	22	32·3
Fair	17	13·2	18	26·5
Poor	19	14·7	16	23·5
	129	100·0	68	100·0

In assessing the trends which appear in the analysis of complications in various groups certain general observations are pertinent.

1. Analysis (Table II) shows that in age-group D (60 years and over) there is a higher percentage of patients with diabetes of long duration as compared with age-group C (40-59 years). It would appear, therefore, that a high incidence of a complication in the older age-groups might equally be due to age or long duration of diabetes. On the other hand, the distribution of the age-groups within each 'duration' group—under 10 years and 10 years and over—is more complex. In the under-10-year group the percentage formed by the ratio of the two younger age-groups combined (25 cases) to the two older age-groups combined (131 cases) is 19·1%. The same percentage in the group with diabetes of 10 years and over is 15·6%. If, however, the composition of the two 'duration' groups is considered with regard to age-groups C and D, there comes to light a much greater discrepancy between the two 'duration' groups, there being a higher proportion of older (Group D) patients in the group with diabetes of longer duration. Therefore differences based on duration of diabetes might well be reflecting the effects of old age.

2. It is necessary to know whether the two racial groups differ materially in respect of duration of diabetes, age, sex, and control. These factors have been studied in the two racial groups and the main conclusions which emerge are as follows:

(*a*) Of the Europeans, 87 (67·4%) had had their diabetes for less than 10 years and 41 (31·8%) for a longer period than this; of the non-Europeans, 69 (85·2%) had had their diabetes for less than 10 years and 11 (13·6%) for a longer period than this. Among the non-Europeans, therefore, there is a higher proportion of patients with diabetes of less than 10 years' duration as compared with the Europeans.

(*b*) If the relationship between race and age is studied in Table II it will be seen that there is a relatively higher proportion of subjects in the younger age-groups among the non-Europeans included in this study.

(*c*) The sex ratio in the two main racial groups is as follows:

Europeans: 49 males, 89 females.
Non-Europeans: 21 males, 60 females.

Thus there is a relatively higher proportion of females in the non-European group.

(*d*) Table I indicates that a somewhat higher proportion of non-European patients fall into the less well controlled groups.

THE RETINAL COMPLICATIONS

It is generally agreed that the earliest stage of diabetic retinopathy consists of punctate micro-aneurysms in the vicinity of the macula. These may be accompanied by small discrete exudates, which may, however, be present alone. Later the exudates enlarge and coalesce to form characteristic irregular, homogeneous, yellowish-white patches. At this stage haemorrhages are present, initially small and round, but subsequently becoming irregular, larger and more numerous.

In the more advanced cases irregular dilatation and tortuosity of veins is seen, coils and loops may be evident, and finally more profuse retinal haemorrhages, vitreous haemorrhages, and the proliferation of newly-formed blood vessels and fibrous tissue, comprise the picture of retinitis proliferans. Subsequent contraction of fibrous tissue may result in retinal detachment.

The occurrence of haemorrhages or aneurysms was noted in 62 patients. In 42 of these, exudates were also present and in 7 additional patients exudates were observed in the absence of haemorrhages or aneurysms. In 3 cases retinitis proliferans was superimposed.

In 69 cases, therefore, diabetes was complicated by the specific retinopathy; 54 were female and 15 male. This

TABLE II. THE RELATION BETWEEN AGE AND DURATION, AND BETWEEN AGE, RACE AND SEX

Age		*Duration*					*Race*					
Group	*Total*	*Under 10 years*		*10 years and longer*		*Ratio + 10 yrs. / − 10 yrs.*	*Europeans*			*Non-Europeans*		
		No.	*% of total under 10 yrs.*	*No.*	*% of total over 10 yrs.*		*M*	*F*	*Total*	*M*	*F*	*Total*
A	12	11	7·1	1	1·9	9·1%	5	1	6	4	2	1
B	20	14	9·0	6	11·5	42·9%	5	4	9	3	8	11
C	83	65	41·7	17	32·7	26·2%	12	35	47	8	28	36
D	95	66	42·3	28	53·8	42·4%	18	49	67	6	22	28
Total	210	156		52			40	89	129	21	60	81

represents a total incidence of 32·9% and an incidence of 44·2% and 24·6% in the females and males respectively.

Separate consideration of the two main racial groups reveals that 37 of the 129 Europeans (21·7%) and 32 of the 81 non-Europeans (39·5%) showed retinopathy.

In respect of the four age-groups defined earlier we find 1 case in Group A, 3 in group B (15·0% of all patients in this age-group), 25 in group C (30·1%) and 40 in group D (42·1%).

In Table III the distribution and incidence with regard to duration of diabetes, insulin requirement and control is shown. In Table IV the distribution according to control of diabetes is separately considered in the two racial groups.

TABLE III. THE RELATION BETWEEN RETINOPATHY, DURATION, INSULIN REQUIREMENT AND CONTROL

	Total No.	No. with retinopathy	Percentage with retinopathy
Duration of diabetes			
Under 10 years ..	156	43	27·4
10 years or longer ..	52	26	50·0
Insulin requirement			
Group 1	54	17	31·5
Group 2	32	23	71·9
Group 3	81	54	66·7
Group 4	43	17	39·5
Control			
Excellent	57	17	29·8
Good	70	18	25·6
Fair	35	11	31·4
Poor	35	19	54·3

TABLE IV. THE RELATION BETWEEN CONTROL, RACE AND RETINOPATHY

Control	Europeans			Non-Europeans		
	Total No.	No. with retinop.	%	Total No.	No. with retinop.	%
Excellent ..	45	11	24·4	12	6	50·0
Good ..	48	14	29·2	22	4	18·2
Fair ..	17	2	11·8	18	9	50·0
Poor ..	19	10	52·6	16	9	73·8

Note. In 4 non-Europeans the degree of control could not be assessed.

Discussion

The first point of interest is that the incidence of retinopathy is higher in the non-Europeans than in the Europeans. As already indicated, the non-Europeans include a relatively higher incidence of patients with diabetes of less than 10 years' duration and a higher proportion of patients under the age of 40 years. Furthermore, separate study shows that, regardless of duration of diabetes (Table V) or age, the incidence of retinopathy is higher in the non-European. If the two main races be studied separately in relation to age the percentage with retinopathy in each age group will be as follows:

European	*Non-European*
Group A: no instances	Group: A 16·7%
Group B: 11·1%	Group: B 18·2%
Group C: 23·4%	Group: C 38·9%
Group D: 37·3%	Group: D 53·6%

The sex differences in the two main racial groups may be of importance, and have been analysed in detail (Table VI). It is apparent that while the European males have a higher incidence of retinopathy than the non-European males, there is a higher incidence of this complication among the non-European females than among the European females. And the high incidence of retinopathy in the non-Europeans as well as the difference in the incidence between the sexes irrespective of race is very largely contributed by this female group. If Table II is examined it will be seen that whereas half the non-European males are under 40 years, only 1/5th of the non-European females fall into this category. It might be supposed that the relatively high proportion of non-European females over the age of 40 years might account for the sex difference in the race group. However, if the incidence of retinopathy among the non-Europeans over the age of 40 years be considered alone, a large difference in incidence can still be demonstrated, viz.: 21·4 and 52·0% respectively for males and females. It may be stated, there-

TABLE V. THE RELATION BETWEEN DURATION, RACE AND RETINOPATHY

Race	Totals		Under 10 years				Over 10 years			
	Race	Retinop.	No.	% of race total	Retinop.	% of race under 10 yrs	No.	% of race total	Retinop.	% of race over 10 yrs.
E	129	37	87	67·4	20	23·0	41	31·8	17	41·5
N-E ..	81	32	69	85·2	23	33·3	11	13·6	9	81·8
All races ..	210	69	156	74·3	43	27·4	52	24·7	26	50·0

TABLE VI. THE RELATION BETWEEN RETINOPATHY, RACE AND SEX

Race	Totals		Males		Females		Ratio males to females
	Race	Retinop.	No.	Retinop.	No.	Retinop.	
E	129	37 (21·7%)	40	11 (27·5%)	89	26 (29·2%)	44·9%
N-E	81	32 (39·5%)	21	4 (19·04%)	60	28 (46·7%)	35·0%
All races	210	69	61	15	149	54	

TABLE VII. THE RELATION BETWEEN RETINOPATHY, AGE AND DURATION

Age-groups	Duration under 10 years			Duration over 10 years		
	Totals	Retinopathy		Totals	Retinopathy	
		No.	Percent		No.	Percent
A	11	1	1/11	1	0	0
	(25)	(1)	(1/25)	(7)	(3)	(42·9%)
B	14	0	—	6	3	50%
C	65	18	27·7%	17	7	41·2%
	(131)	(42)	(32·1%)	(45)	(23)	(51·1%)
D	66	24	36·4%	28	16	57·1%
Total	156	43		52	26	

fore, that whilst non-European females include an unusually large proportion of patients aged 40 years and over, this does not convincingly account for the sex difference in the two races, nor for the difference in incidence between the non-European males and females.

Comments

1. The incidence of retinopathy rises with increasing age.

2. The incidence of retinopathy is higher in the non-European than in the European. This is probably not influenced by age or duration of diabetes but may be associated with poorer control and a large proportion of females in the non-European group. These two features tend to weaken the case for a true racial difference, but suggest a difference in the behaviour of the sexes in the two races.

3. A high incidence of retinopathy in the non-European females compared with European females is not explained by age selection. It appears to account for the racial bias in the group as a whole as well as the difference between the two sexes irrespective of race.

4. If the 'poor' control group is separated from the other groups and the latter are considered together, analysis reveals a very significant difference between the 'poor' control group and the remainder in respect of the incidence of retinopathy, the incidence being higher in the 'poor' control group.

5. No correlation exists between insulin requirement and the incidence of retinopathy.

6. The high incidence in those with diabetes of long duration requires comment, particularly in relation to age selection. The situation is analysed in Table VII, and it is apparent that if the 'duration' groups are considered separately the proportion of patients in age-groups C and D is not very different in the two 'duration' groups, and the incidence of retinopathy rises with age, independent of duration. Among the small number in the younger age-group (A and B) it is interesting to note that of the 7 patients in the over-10-year duration group, 3 (42·9%) had retinopathy, whereas only 1 (4%) of the 25 patients in the under-10-year duration group, developed this complication. All this suggests that both age and duration of diabetes favour the development of retinopathy.

CARDIOVASCULAR COMPLICATIONS

Coronary artery disease was diagnosed in the presence of one or more of 4 features:

1. A history of angina pectoris or cardiac infarction with or without an associated electrocardiographic abnormality at the time of follow-up:

A history of cardiac infarction was obtained in 10 patients, 7 of whom subsequently experienced angina pectoris. In all but one of these there was ECG evidence of ischaemic damage and in this patient the history was acceptable beyond reasonable doubt.

Thirteen patients had angina pectoris only. Of these an ECG was normal in 4, compatible with ischaemia in the remaining 9.

Care was taken to exclude the less usual causes of angina pectoris such as cardiovascular syphilis, severe aortic stenosis, and anaemia.

2. Unequivocal ECG changes of infarction in the absence of a history of ischaemic pain:

Eight patients came into this category. The ECG of 6 additional patients were suggestive of posterior infarction but were possibly within normal limits. These were excluded from the group under discussion.

3. ECG changes suggesting ischaemic damage, i.e. abnormal ST segments and/or T waves, when clinical evidence for an alternative explanation (ventricular hypertrophy, digitalis effect, electrolyte disturbance, etc.) did not exist.

4. Two patients showed left bundle-branch block in the absence of symptoms of heart disease. One was moderately hypertensive, the other was a normotensive European male with symptoms and signs of peripheral vascular disease. These two patients are included in the group with coronary artery disease.

In 45 (21·4% of the total 210) patients these criteria for coronary artery disease were satisfied. They comprised 29 Europeans and 16 non-Europeans (10 Cape Coloured and 6 Malays). This represents an incidence of 22·5% and 19·8% in the two main racial groups.

Sixteen of the patients were male (26·2% of all the males in the series), 29 female (19·9% of all the females).

The age distribution was as follows:

0–19 years	nil
20–39 years	1
40–49 years	6
50–59 years	6
60 and over	32

The incidence in relation to duration of diabetes, insulin requirement and control is tabulated in Table VIII. Unfortunately we have no control series of non-diabetics with

which to compare the incidence of ischaemic heart disease in the cases under review.*

TABLE VIII. THE RELATION BETWEEN CORONARY ARTERY DISEASE, DURATION OF DIABETES, INSULIN REQUIREMENT AND CONTROL

	Total No.	No. with cor. art. dis.	% with cor. art. dis.
Duration of diabetes			
Under 10 years	156	29	12·1
10 years or longer	52	16	30·8
Insulin requirement			
Group 1	54	11	20·4
Group 2	32	10	31·3
Group 3	81	19	23·9
Group 4	43	5	11·6
Control			
Excellent	57	13	22·8
Good	70	18	25·7
Fair	35	6	17·1
Poor	35	8	22·9

Comment

It is found that the incidence of ischaemic heart disease is similar in the two racial groups and in the two sexes. It rises considerably in the older age-group and this may explain the higher incidence in those with diabetes of longer than 10 years' duration.

Some of these features are at variance with certain series in the literature. For example, Warren and Le Compte[10] found that 40% of diabetics have clinical or ECG evidence of coronary artery disease and, of these, 25% are under the age of 40 years.

No correlation appears to exist between the incidence of ischaemic heart disease and insulin requirement or diabetic control.

NEUROLOGICAL COMPLICATIONS

Although specific interrogation included all those complications of diabetes which are believed to have a neurological basis, the emphasis of this analysis will fall on the features that allow objective clinical assessment, which for the most part occur in the lower limbs. In order to separate this group of signs from the other forms of neuropathy it was decided to divide the material into 3 categories as follows:

Group 1. Cases presenting certain common objective criteria in the lower limbs.

Group 2. Cases with leg pain other than intermittent claudication.

Group 3. Other instances of neuropathy not classifiable in Groups 1 and 2.

Group 1

This group provides the corner-stone of this analysis. To qualify for this group there had to be present at least one of the following objective abnormalities:

1. Paresis or paralysis of the lower-motor-neurone type.
2. Tender calves.
3. Absent knee jerk with or without absent ankle jerk.
4. Absent ankle jerk.
5. Grossly diminished or absent vibration sense.

Only those tendon reflexes which were absent after reinforcement were included. Naturally, where these signs had an obvious alternative aetiology they were excluded from the totals.

The material was analysed in respect of incidence, duration, insulin requirement, control, sex, race, age, and in relation to leg pain and evidence of vascular disease.

Total incidence. Of the total of 210 patients in this series, 49 patients exhibited at least one of the above signs, an incidence of 23·3%. In 16 of the 49 patients, more than one of the signs was present, an incidence of 7·6%. The incidence of individual abnormalities within this group was as follows:

1. Paresis of the lower-motor-neurone type—no instances significant to the present survey were found.
2. Tender calves—10 instances, 4·8% of the total in this study.
3. Absent knee jerk—11 instances, 5·2% of the total.
4. Absent ankle jerk—29 instances, 13·8% of the total.
5. Diminished vibration sense—23 instances, 11·0% of the total.

Sex. There were 9 males and 40 females in this group, giving a percentage of the total number of each sex in the series of 14·7% and 27·2% respectively.

Insulin requirement and control. The distribution in respect of these features is shown in Table IX.

TABLE IX. THE RELATION BETWEEN GROUP-1 SIGNS, INSULIN REQUIREMENTS AND CONTROL

	Total in series	No. with group-1 signs	Percent. with group-1 signs
Insulin requirement			
Group 1	54	10	18·5
Group 2	32	5	15·6
Group 3	81	24	29·6
Group 4	43	10	23·2
Control			
Excellent	57	10	17·5
Good	70	16	22·8
Fair	35	7	20·0
Poor	35	14	40·0

Note. In 2 instances the degree of control could not be assessed.

Race. Separate consideration of the two main racial groups reveals that 29 Europeans (22·5% of the Europeans) and 20 non-Europeans (24·7% of the non-Europeans) showed evidence of group-1 neuropathy.

Age. Instances of group-1 neuropathy were distributed among the four age-groups defined earlier as follows:

No instances in age-group A (of 12 patients in this group)
1 in age-group B (of 20 patients in this group)
14 in age-group C (16·8% of the 83 patients in this group)
34 in age-group D (35·8% of the 95 patients in this group)

Because of the obvious relationship between age and duration, the distribution as regards duration in group-C (patients aged 40-59 years) and group-D (those aged 60 years and over) are given (see Table II). Group-C patients with diabetes of 10 or more years duration (17 patients) yielded 3 instances of group-1 neuropathy, an incidence of 17·6%; group-D patients with diabetes of the same duration (28 patients) yielded 9 instances of neuropathy, an incidence of 32·1%. Group-C patients with diabetes

* Dr. B. Bronte-Stewart has kindly made available to us figures from a survey of ischaemic heart disease in the general population in Cape Town. The only group strictly comparable to our own are males (equal numbers of European and Coloured) in the age-group 40-59. The incidence found here was 5·9% (in 239 subjects), which, even though it may tend to underestimate the true frequency, is still very much under the 14·5% shown by our diabetics in this age group. The approximately doubled likelihood of heart disease in diabetics is in accordance with the literature in general.

TABLE X. RELATION BETWEEN NEUROPATHY AND DURATION OF DIABETES

Duration (years)	*Total in series*	*Group-1 Neurop.*		*Group-2 Neurop.*		*Groups 1 and 2 combined*	
		No.	%	*No.*	%	*No.*	%
0– 4	100	18	18·0	13	13·0	31	31·0
5– 9	56	18	32·1	3	5·4	21	37·0
10–14	31	7	22·6	3	9·7	10	32·2
15 + ...	21	6	28·6	3	14·3	9	42·8
	208	49		22		71	

Note. In 2 instances duration was not assessed.

of less than 10 years duration (65 patients) yielded 11 instances of neuropathy, an incidence of 16·9%; group-D patients with diabetes of the same duration (66 patients) yielded 25 instances of neuropathy, an incidence of 37·9%. An increasing incidence of this complication was demonstrable, therefore, regardless of duration. As can be seen in Table II there are quite significant differences in the duration make-up of age-groups C and D, the percentage ratios (longer duration over shorter duration) for these two age-groups being 26·2% and 42·4%.

The incidence of neuropathy in relation to the duration of diabetes is shown in Table X. If the 'duration' groups are combined into 'under 10 years' and 'over 10 years' it will be seen that there were 36 patients with group-1 signs who had had their diabetes for less than 10 years—23·0% of the total number of diabetics in this category—and 13 patients who had had their diabetes for 10 years or more—25·0% of the total in this category.

Group 2

Under this heading are included the several varieties of leg pain, other than intermittent claudication, commonly met with in diabetic subjects. Obviously the assessment of such a symptom presents many difficulties; nevertheless an effort was made to include only genuine instances.

The only points of interest were the incidence of this symptom and its relationship to objective evidence of neuropathy and evidence of vascular disease.

Of the total number of patients only 22 complained of leg pain as defined above, an incidence of 10·5%. In 8 of these patients there were present one or more of the signs included in group 1 (36·4% of the patients with leg pain). Thus, 16·3% of the patients in group 1 had leg pain while the remaining 161 patients in this series yielded only 14 instances of this symptom, an incidence of 8·7%.

Seven patients, or 31·8% of those with leg pain showed evidence of vascular disease in the legs. As will be seen later, there were 48 patients in whom there was evidence of vascular disease in the legs. This gives an incidence of leg pain amongst these patients of 14·6%, as opposed to the incidence of 9·3% among those patients in whom there was no evidence of vascular disease. In 13 of the 22 patients with leg pain there was evidence of either neurological or vascular disease or both, an incidence of 59·1%.

Peripheral vascular disease in relation to neuropathy. Evidence of vascular disease in the lower limbs was sought in each patient on clinical grounds according to whether one or more of the following features were present: Intermittent claudication; absent pulsation in the posterior tibial, dorsalis pedis or other arteries of the lower limbs; amputation because of vascular disease; gangrene; or other obvious signs such as temperature or trophic changes attributable to vascular disease. Vascular disease was present in 48 patients, giving a total incidence of 22·9% for the series. In group 1, 18 patients (36·7% of this group) showed evidence of vascular disease. 37·5% of patients with vascular disease had group-1 signs. Therefore, in 30 patients, or 62·5% of those who had vascular disease, group-1 signs were absent. The 18 patients who presented signs of vascular disease and group-1 neuropathy constituted 8·6% of the total number of patients in this study. 27 Europeans, or 20·9% of the Europeans, and 21 non-Europeans, or 25·9% of the non-Europeans, presented evidence of vascular disease.

Group 3

This group embraces a miscellaneous collection of neurological features not classifiable under groups 1 and 2. Altogether there were 12 instances in this group, 8 of which were associated with group-1 signs, the remaining 4 being present as isolated neurological features.

The majority of examples in this group were instances of sensory disturbance—arm pain, paraesthesiae, superficial sensory loss, etc. There were 2 instances of disturbed bladder function and 1 of external rectus paresis. There were no instances of nocturnal diarrhoea, myelopathy, impotence, or pupillary disturbance.

Discussion

The subject of neuropathy has been adequately reviewed elsewhere.[6,9,7,5,1] It is not proposed, therefore, to review the subject, but merely to draw certain comparisons between our results and those reported in a few major contributions.

Because of the relatively small size of this series of patients it was not possible to draw many conclusions which could be statistically supported or were worth detailed analysis. Nevertheless, a number of trends were demonstrable, some of which are probably highly significant.

Assessment of the *total incidence* of neurological complications attributable to diabetes immediately raises a host of difficulties, each of which adds to the inaccuracy of the figures produced. These difficulties include: Differences in definition, the temporary nature of many features, and the validity of certain symptoms in persons who receive a painful daily reminder that they are abnormal; the problem of isolated neurological deficits, as for instance the absent ankle jerk or diminished vibration sense in relation to advancing age, not to speak of the degree of abnormality which is acceptable (for instance whether a tendon reflex is described as absent or merely diminished); the question whether diabetes is the cause of a neurological sign in a particular instance; and, finally, the limits of what is regarded

as primarily neurological as opposed to primarily vascular.

However, it would appear that the figure of 23·3% for certain common signs in the lower limb or 31·9% for all forms of neuropathy, both subjective and objective, arrived at in this survey is well within the wide range suggested by Bailey.[1] In his review Bailey found that, if all forms of neuropathy were included, the clinical incidence appeared to be approximately 50%, whereas he estimated that clinically significant diabetic neuropathy was probably less than 5%. Indeed, the incidence given by various workers has differed much more widely than this.[1,5]

Turning to a consideration of the *group*-1 signs, i.e. certain common signs occurring primarily in the lower limb, it is possible to make the following remarks. Of the *individual defects* the absent ankle jerk and loss of vibration sense were by far the commonest signs elicited. These had an incidence of 13·8% and 11·0% respectively of the total number of patients in the series, or 59·2% and 46·9% of the 49 cases in group 1.

It is generally agreed that absent deep tendon reflexes and diminished vibration sense are the commonest signs of diabetic neuropathy.[5,3,4,9,2] Reports in the literature give a variety of results for the incidence of these signs in the general diabetic population and for the relative incidence of these signs in groups of people showing evidence of neuropathy. Amongst their diabetic subjects Goodman *et al.*[5] found the ankle jerk to be absent in 48·2% and the knee jerk in 25·6% of subjects; amongst his 125 cases of diabetic neuropathy, Rundles[9] found that 81·0% had absent ankle jerks and 56·0% had absent knee jerks. The approach of different workers to these signs has varied. This is best illustrated by the wide differences which exist in the reports of diminished or absent vibration sense. There is general agreement that impaired vibration sensibility is the earliest and often the only sign of diabetic peripheral neuropathy. In Rundles' series of 125 cases with diabetic neuropathy, vibration sense was diminished or absent in 45·6%, a point of agreement between our study and his inasmuch as he found gross involvement of vibration sense to be less common than loss of tendon reflexes in the lower limbs. We feel that obvious impairment of vibration perception as tested by ordinary clinical methods is of some value in the estimation of what Bailey[1] has termed 'clinically significant diabetic neuropathy'. The probable exaggeration of its incidence as a result of non-specific factors, particularly age, should, however, be clearly appreciated.

Although at first glance there appeared to be a striking *sex difference* with respect to group-1 signs, an incidence of 26·8% and 14·7% respectively of the totals for female and male, more detailed analysis in relation to age (see Table II) and control revealed significant differences in the distribution of these two factors, particularly age, in the two sex groups. Separate consideration of the two sexes in respect of the two broad age-groups under and over 40 years demonstrated that age selection had some influence on the incidence in the two sexes in this analysis, the difference being due in certain measure to the significantly higher proportion of males in the younger age-group.

The table (Table IX) dealing with the occurrence of group-1 signs in respect of *duration*, *insulin requirement* and *control* details some interesting trends. Duration, if considered in two large groups—under 10 years and 10 years or longer—did not effect the incidence of these complications. When this analysis was carried further, into 5-year groups (see Table X) the effect of duration was still not dramatic, although some influence was demonstrable. The picture varied according to whether groups 1 and 2 were considered separately or combined. Of some interest was the relatively high incidence of neuropathy in the under-5-year group as well as the relative frequency of leg pain at the two extremes of duration of our study.

Insulin requirement did not influence the incidence of group-1 signs and in this respect our findings are in agreement with those of Jordan[6] as applied to his 'neuritic' group.

Analysis of control, however, provided an interesting result as seen elsewhere in this paper in relation to retinopathy. If the incidence in the 'poor' control group is contrasted with the incidence in the remaining 3 groups combined there results a highly significant difference between the two groups, there being a far higher liability to group-1 signs in the poorly controlled diabetic in our series. In case this reflected some gross difference in chance selection of other possibly influential factors the distribution of such factors as age, duration and sex was analysed; these features were found to be roughly comparable in the two groups.

Most authors would agree that whereas control is strongly correlated with the development of neuropathy[5,7,4] yet 'it is likely that neither the amount of dextrose in the blood nor the acetone and diacetic acid are the actual toxic agents in producing neuropathy'.[5]

Whilst it is well known that there is a variable relationship between duration of diabetes and the onset of neuropathy, there being instances in which symptoms have ante-dated the diabetes,[6,1] it is generally agreed that a period of somewhat over 5 years,[7,6,9,1] or longer,[2] usually precedes the onset of neurological features and that there is clear indication of the importance of duration.[5]

With regard to *race*, this survey showed no significant difference in liability to neurological complications between the two main racial groups, i.e. European and non-European.

Age appeared to exert considerable influence on the incidence of this limited group of neurological signs inasmuch as the incidence in the oldest group (group D) was twice that in the next group (group C). Numbers in the younger groups were too small to enable any comparison with these, although it is possibly worth mentioning that only one case was found under the age of 40 years.

Leg pain, as defined here, was revealed to be less frequent than anticipated if the incidence of 10·5% in this study is compared with the incidence derived from other sources. Often this feature is referred to by writers as 'the commonest symptom' of diabetic neuropathy without further attempt being made to derive its actual incidence among the diabetic population or to relate its incidence to the other features of diabetes. Where figures for pain in the extremities have been given they have varied from 5% to 49%, most authors giving an incidence above 10%.[5] As might be expected, analysis of the interrelationship between leg pain, neurological disorder in the legs, and signs of vascular insufficiency, underlined the alliance between these three aspects, not that any confirmation of an aetiological relationship can be assumed from these facts. Roughly one-third, or 36·4% of the patients with leg pain had one or more of the group-1 neurological signs, and roughly one-third, or 31·8%, pre-

sented signs of vascular insufficiency. If, however, the incidence of these three factors is combined, it can be shown that over one-half, or 59·1%, of patients with leg pain will have either neurological or vascular disorder, or both. Looking at this situation from another point of view, the incidence of leg pain among those patients with group-1 signs and among those with vascular disorder was roughly double that in the remainder of the patients under investigation. Despite these relationships, the large number of instances of leg pain unexplained on clinical grounds by the coexistence of neurological or vascular disorders enhances, at least in some measure, the probability of a functional metabolic mechanism in many cases.

Study of the *relationship between group-1 neurological signs and vascular disease* suggested a certain parallel between the development of these two disorders. The incidence of neurological disorder in the vascular group (37·5%) and the incidence of vascular insufficiency in group-1 (36·7%) were both significantly higher than the incidence of the corresponding disorders in the total of our patients, 23·3% and 22·9% respectively for the neurological and vascular groups.

No racial differences were detected in the incidence of vascular insufficiency in the lower limbs.

The alliance between these two disorders is probably largely conditioned by the fact that both have their maximum incidence in the older diabetics. It should be stressed that well over half of the group-1 patients had no evidence of vascular disease, and therefore, whatever the aetiological relationship between the two from a histological point of view, clinical support for such contentions would appear far from established in many, if not most, instances of diabetic neuropathy.

Evidence of occlusive peripheral vascular disease in diabetic subjects appears to vary widely. Examples of the incidence of occlusive vascular disease among diabetics given in the literature are 46·1%[5] and 37·0%.[8] As to the overlap between neuropathy and occlusive vascular disease Goodman *et al.*[5] in two groups of cases found the incidence of peripheral disease among those diabetics with neuropathy to vary from 36·4% to 54·7%, thereby leading that group of workers to the conclusion that whereas there was an obvious relationship between the two disorders the correlation was not yet sufficiently strong to establish a definite aetiological role.

Comment

The total incidence of neurological disorders arrived at in this study agrees with that group of authors who have made rather more conservative claims for the incidence of these complications. This fact has resulted partly from the use of simple clinical methods only, from obedience to unequivocal criteria, and from the fact that this study was confined to out-patients. It is felt that this approach, being widely applicable, has given results which will be of use for comparative purposes in large-scale investigations. Moreover, it allows possibly a closer estimate of what may be termed 'clinically significant' diabetic neuropathy.

Of the various aspects which were investigated, the most significant influences appeared to be increasing age and poor control. Our results demonstrate a parallel between clinically evident vascular insufficiency and neurological signs in the lower limbs, although most instances of neuropathy were not involved in this relationship.

Of the individual signs in the lower limbs, absent ankle jerks and diminished vibration sense were found more commonly than other signs. These two signs were found more commonly than objective evidence of significant superficial sensory loss.

There was some alliance between leg pain, as defined, and the presence of neurological and vascular disease, but in a large number of patients the occurrence of pain was not accompanied by clinically detectable organic disease.

CONCLUSIONS AND SUMMARY

The retinal, cardiovascular and neurological abnormalities occurring in 210 consecutively examined diabetic out-patients have been analysed. The patients are of mixed racial origins, and include white 'Europeans', Cape Coloured subjects, Cape Malays and Bantu. The incidence of clinically apparent renal disease was too small for analysis.

In general, all these 'complications' showed a tendency to increase with age; as regards duration of the diabetes, the neuropathy showed no such trend, while the incidence of vascular disorders did increase. There was no correlation between insulin dosage and incidence of complications, but most interesting was the finding of a significantly higher rate of retinopathy and of neuropathy in the 'poor control' group, whilst in cardiovascular disease the control of the diabetes appeared to play no part.

'White' and 'non-White' diabetics showed little or no difference in the incidence of the complications under discussion, except that retinopathy was unexpectedly frequent in Coloured females, even allowing for the higher proportion of these patients in the 'poor control' group. It must be realized that the pure Native (Bantu) diabetics were not considered separately, and none of this discussion applies to them as a race.

We should like to thank all others who were associated in this survey, including particularly Prof. G. C. Linder, Dr. Anne Linder, Dr. V. Schrire, Mr. John Daragh (statistician), Miss M. Rom (typist), and the ophthalmologists. The Medical Superintendent of Groote Schuur Hospital and Prof. J. F. Brock encouraged its inception and continuation, and Prof. F. Forman commented upon the manuscript. Finally, we acknowledge the financial assistance of the Council for Scientific and Industrial Research by grants to the Endocrine Research Group.

Note. Full statistical analysis has not been included, but is available on request from Dr. E. A. Allen.

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POLYARTERITIS NODOSA: TWO CASES AND A REVIEW

NEVILLE BECKER, M.B., CH.B., *Addington Hospital, Durban*

Polyarteritis nodosa, though still a rare disease, is becoming more and more common. Credit is usually given to Kussmaul and Maier of Germany for the first description of the condition in 1866.[1] By 1914 only 5 cases had been reported in the USA; today hundreds of cases can be collected in the world literature.

The aetiology has remained obscure. Amongst others, Rich and Gregory[2] have stressed the importance of hypersensitivity as an aetiological factor. Selye had postulated that psychogenic factors, acting through the adrenal cortex, may also be a causal factor.[2] Rich[3] reported polyarteritis nodosa in a number of cases treated with sulphonamides. The increasing incidence of the collagen diseases, on the assumption that hypersensitivity and the antigen-antibody reaction are the primary aetiological factors, becomes explicable by the wider and freer use of the drugs, particularly serum, sulphonamides and penicillin. According to Rich and Gregory[2] these drugs are most frequently associated with, if not related to, the development of polyarteritis nodosa.

The following two cases of polyarteritis nodosa, both female, were admitted to the same ward (of only 30 cases) within less than a month of each other.

CASE 1. POLYARTERITIS NODOSA PRESENTING AS CARCINOMA OF THE LUNG

Mrs. M.S., aged 61, was admitted on 13 April 1956 complaining of sudden onset of fever, dyspnoea, haemoptysis, loss of appetite and a feeling of weakness, 7 days before admission. According to her husband, however, she had not been well for the last few months. She had been told she had influenza and given antibiotics without response. There was a history suggestive of a coronary episode 4—5 days before admission. She also complained of left-sided headaches for the last 4 years. There was no history of previous illnesses or of tuberculosis in the family.

Examination

The general examination was almost entirely negative, the only points of interest being (1) pyrexia of 100°F, (2) chest expansion greater on the left side, and (3) general wasting.

Urine: Albumen—trace. Red blood cells ++.

Blood: Haemoglobin 11·1 g.%. White blood cells 28,000 per c.mm. (92% of polymorphs).

Chest X-ray: On the right, (*a*) well-defined round area of consolidation in mid-zone, (*b*) apical fibrosis and consolidation posterior upper lobe, and (*c*) hilar and basal calcification. On the left, basal fibrosis.

ECG showed evidence of a recent and an old infarct.

Course in Hospital

During the stay in hospital the temperature was of swinging character, the highest temperature recorded being 102°F. The patient became extremely apathetic 5 days after admission, when the serum electrolytes per litre were: Na 131 mEq., Cl 93 mEq., K 5·5 mEq., CO_2 combining power 27 vols.%. The blood urea was 50 mg.%.

The chest X-ray looked like carcinoma of the lung. A thoracic surgeon was called in and it was decided to resort to bronchoscopy to elucidate the pathology. Bronchoscopy was abandoned because the patient had deteriorated very rapidly and it was found on further investigation that the blood urea had risen to 305 mg.%. Two days later, on 26 April, the patient died—13 days after admission.

Post-mortem Examination

Autopsy showed (1) consolidation of middle zone of right lung and left and right apices, thought to be tuberculous lesions, (2) myocardial infarction, both old and fresh, (3) almost complete infarction of the spleen, and (4) acute bilateral pyelonephritis.

On microscopic examination the heart, spleen, kidneys and lungs all showed necrotizing vascular lesions characteristic of periarteritis nodosa, the lung lesions being of a more chronic nature than those in the other organs.

Discussion

At the time of admission a diagnosis of pulmonary tuberculosis was favoured, until the chest X-ray presented an appearance more like that of carcinoma of the lung. The symptoms, though apparently acute on onset, would have passed for either condition. A diagnosis of carcinoma of the lung was then held clinically, the temperature and leucocytosis being assigned to secondary infection of the carcinoma.

The thoracic surgeon who was consulted agreed that the case looked like carcinoma of the lung, but before bronchoscopy could be performed the patient deteriorated rapidly and died in uraemia.

Pulmonary Involvement

Haemoptysis is a rare finding in this disease; it was recorded in only 1 of 243 cases of polyarteritis nodosa reviewed by Harris, Lynch and O'Hare.[4]

Miller and Daley[5] describe the lung involvement and its radiological aspects. The basic pathology appears to be perivascular infiltration, oedema and haemorrhage; it has been likened to thrombo-angiitis obliterans by Harkavy.[6] Although a miliary appearance, opacities of moderate density, pulmonary infarctions, pleural effusions and atelectasis may occur; the classical picture is one of a fan-like pulmonary infiltration due to increased vascular markings extending out from the hilar region.

Polyarteritis nodosa seems to spare the periphery of the lung, yet in this patient both apices were involved. The chest X-ray in this case was unusual for polyarteritis nodosa, and even at autopsy the provisional diagnosis (until microscopic examination) was tuberculosis, with the reservation of possible alveolar carcinoma or visceral angiitis, which alone could have explained the multiple organ involvement.

Other Manifestations

Besides the lung involvement there are further interesting aspects in this case:

Myocardial infarction is a rare finding in polyarteritis nodosa.[5] There is no characteristic ECG pattern, but flattening of the T waves and left ventricular strain are sometimes found. In this case the ECG showed evidence (confirmed *post mortem*) of both an old and a recent infarct. Microscopically lesions of polyarteritis nodosa were demonstrated in the heart.

Splenic infarction is found *post mortem* in less than a third of cases. This case showed gross infarction of the spleen. No thrombus was demonstrated in the splenic vessels.

Renal involvement and uraemia. The kidneys are involved in 70—80% of cases of polyarteritis nodosa, yet strangely enough clinical uraemia was observed in only 13% of a series of 177 cases.[4] This patient died in uraemia with a blood urea of over 300 mg.%.

CASE 2. POLYARTERITIS NODOSA WITH ANURIA THE PRESENTING FEATURE

Mrs. K., aged 44 years, was admitted on 1 May 1959 with the following complaints:

(1) Continuous backache and lower abdominal pain for 2 weeks. (2) Sensation of a lump in the throat for 10 days. (3) Itchy skin for 7 days. (4) Pins-and-needles and stiffness of hands and feet for 4 days. (5) Urinary output almost *nil* for 3 days, with anorexia and vomiting. (6) Poor vision in the right eye for 10 years, and in the left eye for 1 year.

The reason why the patient was sent to hospital was her anuria. She had only passed a cupful of urine in 3 days, and catheterization did not produce more than a few drops. The accompanying vomiting and anorexia were severe.

Past Illnesses

1946, blurring of vision of right eye (diagnosed as choroiditis). 1948, cholecystitis (refused operation). 1952, had 'yellow jaundice' n Johannesburg (given intravenous cortisone). 1953, further attack of jaundice—again given cortisone. Badly swollen legs and abdomen, with dyspnoea, later in 1953; also headaches for an indefinite period. 1955, pyelitis with severe backache similar to present backache; later in the year, dysentery and vomiting.

Examination

Pulse 100 per minute. Blood pressure 140/90 mm.Hg. Temperature normal. Soft systolic murmur over all areas. Varicose veins in both legs, with incompetent perforators. Firm 5-finger hepatomegaly. Fundi—vessels slightly tortuous on left side, with pale area on choroid. Ankle jerks absent.

Urine: Albumen—trace. Red blood cells ++. 4 pus cells per high-power field. On ureteric catheterization, right pelvis—albumen trace, red blood cells +++, few pus cells; left pelvis—albumen +++, red blood cells ++, more than 15 pus cells per high-power field, *B. coli* isolated.

Blood: Haemoglobin 9·2g.%. White blood cells 6,000 per c.mm. Differential count normal. Serum electrolytes (mEq. per litre)—Na 150, K 5·7, Cl 105·2. CO_2 combining power 24 vols.%.

Liver functions: Bilirubin 0·6 mg.%. Proteins 6·8 g.%, albumen-globulin ratio 1·3 : 1. Prothrombin index 92%.

Kidney functions: Blood urea 76 mg.% on admission; rose steadily to 123 mg.% after 2 months. PSP test—only 10% clearance after 2 hours.

Bone-marrow: Slight decrease in erythropoiesis. No L.E. cells.

X-rays: Chest normal. Three intravenous pyelograms showed no dye excretion after 1 hour. Retrograde pyelograms normal.

Biopsies: Liver—fatty change, increased fibrous tissue, periportal round-cell infiltration, and evidence of regeneration. Subcutaneous nodule—characteristic appearance of polyarteritis nodosa.

Course in Hospital

Soon after admission the patient commenced putting out good amounts of urine and the urinary output remained good throughout her course. The blood pressure rose from 140/90 mm. Hg. on admission to 170/100 after a few days, and remained more or less at this level. The ophthalmologist reported central scotomata in both eyes, old choroiditis in the left eye, early hypertensive retinitis in the left eye, and old exudates in both discs.

While in hospital the patient developed small subcuticular haemorrhages in her legs, arthritis of the left knee with effusion, and a few small subcutaneous nodules (biopsy characteristic of polyarteritis nodosa—see above).

Termination. The patient died about 3 months after her discharge from hospital in July 1956. She had been on cortisone therapy.

Discussion

The chief presenting symptom in this case was the low urinary output, which amounted to anuria (under 100 cc. daily) for 3 days. What little urine was obtained was loaded with red blood cells and albumen, but no casts were present.

Anuria. Although anuria as a presenting symptom in polyarteritis nodosa has been recorded in the literature (Rolnick and Davidson[8]), it is comparatively rare. The two main pathological conditions affecting the kidneys are haemorrhage and infarction and glomerulonephritis, the former in 55% of cases, the latter in 33%. Clinically this patient was not affected with glomerulonephritis. An interesting possibility is a massive infarction resulting in bilateral cortical necrosis; such a case was recorded by Wordley.[9]

Other Manifestations

Besides the anuria there are further interesting aspects in this case:

Hepatomegaly and jaundice. It is difficult to ascertain the significance of the hepatomegaly since this patient in 1952 and 1953 had two attacks of jaundice, which might have been due to infective hepatitis, and this might have left cirrhosis and hepatomegaly. Jaundice itself, however, has been reported in 12% of the 177 cases reviewed by Harris, Lynch and O'Hare.[4] It is quite possible that this jaundice was a manifestation of the polyarteritis nodosa. Hepatomegaly itself is not an uncommon finding in polyarteritis nodosa; it is associated with infarctions in the liver.

Visual disturbance. Choroiditis was diagnosed in hospital in this patient's left eye. Visual disturbance in this eye has been present for only 1 year. In contrast to this, poor vision has been present in her right eye for 10 years. There is a history of trauma over the right eye just before visual disturbance occurred, and it seems unlikely that polyarteritis nodosa would have caused this poor vision in 1946, although it probably did cause the choroiditis in the left eye 1 year ago.

Peripheral neuritis. This patient complained of pins-and-needles and stiffness of hands and feet and the ankle jerks were found to be absent. Gruber[10] reported a high incidence of peripheral neuritis in polyarteritis nodosa, and other series tend to confirm this. The basis of the neuritis appears to be involvement of the nutrient arteries to the nerves, resulting in ischaemia and degeneration.

Anaemia, subcutaneous nodules, arthritis, hypertension and subcuticular haemorrhages are all fairly common findings in polyarteritis nodosa. They occurred in the following percentages in the series of 177 cases reviewed by Harris, Lynch and O'Hare:[4] Anaemia 66%, hypertension 53%, arthritis 34%, petechiae and purpura 27%, and nodules 23%.

SUMMARY

Two cases of polyarteritis nodosa are reported on and compared with a review of the literature. Both females, they presented within less than a month in a ward of only 30 patients.

The fulminating character of polyarteritis nodosa in case 1 contrasts with a more chronic picture in case 2.

In the acute case the diagnosis was only established *post mortem*. In case 2 the diagnosis was made *ante mortem*.

I should like to express my thanks to Dr. J. V. Tanchel, Superintendent, Addington Hospital, Durban, and Dr. B. Moshal, whose cases these were, for allowing me to publish this report. My particular thanks to Dr. A. Lurie for much advice in preparing this article.

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THE COLLEGE OF PHYSICIANS, SURGEONS AND GYNAECOLOGISTS OF SOUTH AFRICA
DIE KOLLEGE VAN INTERNISTE, CHIRURGE EN GINEKOLOË VAN SUID-AFRIKA

TRAVELLING FELLOWSHIP IN PSYCHIATRIC MEDICINE

The first travelling Fellowship in Psychiatric Medicine has been awarded by the College of Physicians, Surgeons and Gynaecologists of South Africa to Dr. Morris Ginsburg, Physician Superintendent, Sterkfontein Hospital, Krugersdorp.

This Fellowship, valued at £1,000, is offered by the College on alternate years beginning in 1960. It has been made possible through the generosity of an anonymous donor. It is the wish of the donor that the award should be made to an outstanding South African psychiatrist who is likely to benefit from first-hand experience of recent advanced psychiatric work being carried out in other countries, with a view to his being able to give his colleagues and his students the advantage of his experience on his return to the Union.

Dr. Morris Ginsburg graduated at the University of Cape Town in 1936 and entered the Mental Hospital Service the following year. From 1937 to 1948 he served on the staffs of various mental hospitals throughout South Africa and in December 1948 he was appointed Assistant Superintendent to Town Hill Hospital, Pietermaritzburg. Subsequently, he was Physician Superintendent of the Alexandra Institution, Maitland; the Oranje Hospital, Bloemfontein; Fort Napier Hospital, Pietermaritzburg; and in January 1959 he was appointed Physician Superintendent of Sterkfontein Hospital, Krugersdorp, which is the teaching hospital of the Medical School of the University of the Witwatersrand.

In 1951 Dr. Ginsburg obtained his M.D. degree, *cum laude*, at the University of Pretoria and he has acted as external examiner in psychiatry to the Universities of Pretoria and Natal. He is an active member of the Executive Committee of the South African National Council for Mental Health and, despite his onerous administrative duties, he is actively engaged in teaching and research. For the past 2 years he has been a member of the Executive Committee of the Faculty of Neurology and Psychiatry of the College of Physicians, Surgeons and Gynaecologists of South Africa and Vice-president of the Association of Neurologists, Psychiatrists and Neurosurgeons (M.A.S.A.).

While overseas Dr. Ginsburg will study recent advances in the treatment of mental illness and administrative methods, as well as modern architectural developments in the design of psychiatric hospitals and early treatment centres. He will also pay special attention to advances in child psychiatry in the field of promotive mental health. In addition, he will undertake a survey of the law as applied to mental illness in the countries he visits, in view of the possible amendments likely to be made in the Mental Disorders Act of the Union of South Africa. Institutions for the treatment of psychopathic personalities in Europe and America will be included in his itinerary.

Dr. Morris Ginsburg is married and has 3 children. His wife is a practising psychiatrist and his son is in his final year of medicine at the University of Cape Town. Dr. Ginsburg expects to leave for Europe early in January 1960.

OFFICIAL ANNOUNCEMENTS : AMPTELIKE AANKONDIGINGS

MEDICAL ASSOCIATION OF SOUTH AFRICA : MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held in St. Saviour's Hall, St. Peter's Road, East London, on Thursday 24 September 1959, at 9.30 a.m.

Agenda

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the Minutes.
5. Financial statement by Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from previous meeting.
9. Notices of motion transferred from previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin
Secretary

Medical House
Cape Town
3 August 1959

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word in die St. Saviours-saal, St. Petersweg, Oos-Londen, op Donderdag 24 September 1959, aanvang 9.30 vm.

Agenda

1. Kennisgewing wat die vergadering belê.
2. Volmagte.
3. Notule van die vorige vergadering (reeds uitgestuur).
4. Sake wat uit die notule voortspruit.
5. Finansiële verslag van die Ere-penningmeester.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae van vorige vergadering oorgehou.
9. Voorstelle waarvan kennis op vorige vergadering gegee is.
10. Nuwe kennisgewings van voorstelle.
11. Ander sake.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
3 Augustus 1959

ANNUAL GENERAL MEETING

Notice is hereby given that the Annual General Meeting of the Medical Association of South Africa will be held in St. Saviour's Hall, St. Peter's Road, East London, on Thursday 24 September 1959, at 12.30 p.m.

Agenda

1. Minutes.
2. Annual Report and Balance Sheet.
3. Election of Auditors.
4. Induction of President.
5. Other business.

At the conclusion of business, the meeting will be adjourned and reconvened at 8 p.m. on Monday 28 September 1959, in the City Hall, East London, where, combined with the Opening

ALGEMENE JAARVERGADERING

Kennis geskied hiermee dat die Algemene Jaarvergadering van die Mediese Vereniging van Suid-Afrika gehou sal word op Donderdag 24 September 1959, om 12.30 nm., in die St. Saviours-saal, St. Petersweg, Oos-Londen.

Agenda

1. Notule.
2. Jaarverslae.
3. Verkiesing van Ouditeure.
4. Inleiding van President.
5. Ander besigheid.

Na afhandeling van dié werksaamhede, word die vergadering verdaag en op Maandag 28 September 1959, om 8 nm. in die Stadsaal, Oos-Londen, hervat, wanneer die openingsplegtigheid

Ceremony of Congress, the President will deliver his Presidential Address. Academic dress will be worn.

A. H. Tonkin
Secretary

Medical House
Cape Town
3 August 1959

van die Kongres plaasvind en die President sy voorsittersrede sal lewer. Akademiese drag word gedra.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
3 Augustus 1959

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Gerald M. Lurie, paediatrician, will move into new rooms at 105 Medical Centre, Heerengracht, Cape Town, on 1 September. Telephones: Rooms 3-1002, residence 44-9433; if no reply 7-2404.

* * *

Dr. Raymund Theron, M.R.C.S. (Eng.), M.D. (Londen), van Bloemfontein, het sy adres verander na Medfontein 115, St. Andrewstraat, Bloemfontein. Telefone: Spreekkamer 2574, woning 3643.

* * *

Association of Physicians of South Africa (M.A.S.A.). At a recent meeting of the Committee of the Cape Town Sub-group of this Association it was unanimously agreed that the name of the Sub-group should be altered to the *Cape Western Sub-group* to include those physicians who live and practise outside the Cape Town area.

* * *

Mr. G. S. Muller Botha has commenced practice as a thoracic surgeon at 502 Medical Centre, Heerengracht, Cape Town. Telephones: Rooms 3-1717, residence 5-5251.

Dr. G. S. Muller Botha het begin praktiseer as borschirurg te Mediese Sentrum 502, Heerengracht, Kaapstad. Telefone: Spreekkamer 2-1717, woning 5-5251.

* * *

Dr. J. H. Hofmeyr, oor-, neus, en keelarts, het na 'n uitgebreide studiereis in Europa en Brittanje weer sy praktyk hervat te van Riebeek Mediese Gebou 37, Schoemanstraat, Pretoria. Telefoon 3-5211.

Dr. J. H. Hofmeyr, otorhinolaryngologist, has now resumed his practice at 37 van Riebeek Medical Buildings, Schoeman Street, Pretoria, after an extended study tour of Britain and the Continent.

* * *

South African Society of Obstetricians and Gynaecologists (M.A.S.A.). The Southern Transvaal Sub-group are organizing the 9th Interim Congress of Obstetrics and Gynaecology in Johannesburg from 20 to 23 April 1960. Interested doctors are requested to prepare and submit papers for the Interim Congress to the local Group Secretary. A detailed programme will be published in a later number of the *Journal*.

* * *

Cape Western Branch, Northern Areas Division (M.A.S.A.). The August meeting of this Division will be held in the Banqueting Hall, Civic Centre, Voortrekker Road, Parow, on Thursday 20 August at 8.15 p.m. Dr. H. O. Hofmeyr will speak on 'Impressions of medicine in the USA'. Refreshments will be served and interested practitioners from other Divisions are welcome. (Telephone 98-8461/3.)

At this meeting an amendment to Rule 38 of the Cape Western Branch (Standing Committees) will be discussed.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Tuesday 18 August at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Dr. Barney Kaplan will speak on 'Malnutrition, pre-pellagra and kwashiorkor with special emphasis on clinical signs in various communities'. All who are interested are invited to attend this meeting.

* * *

Dr. O. V. S. Kok van Pretoria, wat tot onlangs Senior Lektor en Hoof was van die Departement Anestesiologie aan die Universiteit van Pretoria, is onlangs bevorder tot professor in hierdie vak. Dit is 'n belangrike gebeurtenis aangesien professor Kok die eerste persoon is aan wie 'n professoraat in die anestesiologie in Suid-Afrika toegeken is. Namens die lede van die Mediese Vereniging wil die *Suid-Afrikaanse Tydskrif vir Geneeskunde* hom gelukwens, in sy hoedanigheid as lid van die Vereniging sowel as in sy kollegiale hoedanigheid van mederedakteur van *Geneeskunde*.

* * *

Die Nuwe Mediese Sentrum, Medfontein, Bloemfontein, was 'n geruime tyd al in die aanbou. Die sentrum is enig van sy soort in die land omdat dit uitsluitlik aan dokters behoort en deur hulle beheer sal word. Die volgende is 'n lys van die dokters wat spreekkamers in die sentrum gaan betrek:

C. Albertyn	D. J. Franck	R. J. Tahan
P. H. L. Barker	M. J. Goddefroy	J. P. Theron
D. J. J. Bezuidenhout	W. Grundill	R. Theron
G. C. Borggreve	A. W. Jacobsz	J. G. Thomson
R. W. Busschau	J. F. Krige	C. V. du Toit
D. C. J. Carter	N. H. J. Louw	J. S. Visser
P. Connan	J. D. Meyer	A. J. Vorster
R. S. Deane	A. G. M. Morrison	J. S. van der Heever
B. de Villiers	J. G. Muller	C. V. van der Merwe
P. E. Dreyer	L. H. Muller	J. W. van der Riet
T. B. Enslin	S. W. Nolte	J. H. J. van Vuuren
Von W. Eybers	F. P. Scott	M. H. Wessels
P. M. S. Fischer	G. W. Snyman	

* * *

College of General Practitioners, Cape of Good Hope Faculty. At an informative General Meeting held on 21 July the proposed functions of the recently formed Faculty were enthusiastically discussed. The main topic under discussion was postgraduate study, and various suggestions, particularly from country practitioners, were made which would facilitate their efforts in keeping abreast of recent advances in medicine. Tape and gramophone recordings of lectures were thought to be of great advantage to practitioners in isolated communities, and lectures and demonstrations by members of the teaching staff of the universities would be of benefit to urban general practitioners.

Undergraduate education in general practice was discussed, and it was decided to investigate the possibility of final-year students being attached to country hospitals for short periods to give them an idea of country practice. Other schemes were also discussed.

Research work in general practice is costly and it was hoped that group research projects would be more fruitful at this stage than individual efforts.

It was reported that the possibility of forming Faculties o. the College in Port Elizabeth, Pretoria, and in Natal was being mooted, and it was felt that once three Faculties had been established a separate union should be formed as had proved so successful in Scotland, Australia and New Zealand.

Kollege van Algemene Praktisyns, Fakulteit Kaap de Goede Hoop. Op 'n vergadering wat op 21 Julie gehou is vir die bespreking van algemene sake, is die voorgestelde doelstellings van die Fakulteit wat onlangs gestig is entoesiasties bespreek. Die hoofonderwerp van bespreking was nagraadse studie en verskeie aanbevelings is gemaak, veral deur plattelandse praktisyns, oor wat gedoen kan word om hulle te help om op die hoogte van sake te bly ten opsigte van nuwe ontwikkelinge in die medisyne. Band- en grammofoonopnames van lesings kan moontlik van groot waarde wees vir praktisyns in afgesonderde gemeenskappe, en demonstrasies deur lede van die onderwyspersoneel van die universiteite kan baie beteken vir stedelike praktisyns.

Opvoeding van ongegradueerdes vir die algemene praktyk is bespreek en dit is besluit om die moontlikheid te ondersoek om dit vir finale-jaar studente moontlik te maak om kort tydjies by plattelandse hospitale te werk om hulle 'n idee te laat kry van die plattelandse praktyk. Ander skemas is ook bespreek. Navorsingswerk in die algemene praktyk is duur en daar word gevoel dat projekte vir navorsing deur 'n groep meer vrugbaar sal wees op hierdie stadium as indiwiduele pogings.

Kennis is geneem daarvan dat die moontlikheid van die stigting van fakulteite van die Kollege in Port Elizabeth, Pretoria, en in Natal oorweeg word, en daar word gevoel dat as hierdie drie fakulteite eers gestig is, 'n aparte unie gevorm sou kon word—wat so suksesvol geblyk het te wees in Skotland, Australië en Nieu-Seeland.

* * *

The Maurice Weinbren Award in Radiology. 1. This Award consists of a certificate and a prize to the value of £25.

2. It will be made annually (in respect of a calendar year) for a published paper of sufficient merit dealing either with radiodiagnosis or radiotherapy.

3. The Award is restricted to medical practitioners registered in South Africa, but the paper may have appeared in any medical journal published in South Africa, or in the *British Journal of Radiology* or the *Journal of the Faculty of Radiologists*, London.

4. The Selection Committee may change or add to the names of the journals in which candidates may have published papers submitted for consideration.

5. Authors who wish to be considered for this Award must advise the Honorary Secretary of the Selection Committee to this effect by 31 December each year.

6. They must provide 6 copies of the paper submitted for consideration not later than the end of February in the succeeding year.

7. The Selection Committee consists of: Prof. S. F. Oosthuizen; Dr. Harris Jackson; Dr. M. H. Fainsinger; Dr. T. Fichardt; Dr. J. N. Jacobson, and Dr. H. A. Shapiro (Acting Honorary Secretary).

The address of the Acting Honorary Secretary is: P.O. Box 1010, Johannesburg.

8. Members of the Selection Committee are not eligible for the Award.

9. The decision of the Selection Committee, in connection with the making of an Award, is final and binding.

* * *

Die Suid-Afrikaanse Akademie vir Wetenskap en Kuns het gedurende die week 24-29 Julie sy 50-jarige bestaan gevier te Stellenbosch. Die Tak Simon van der Stel van die Afdeling Geneeskunde van die Akademie het in hierdie verband 'n vergadering gereël op 23 Julie. Prof. M. W. Woerdeman, die bekende histoloog-anatoom van die gemeentelike Universiteit van Amsterdam en voorsitter van die Koninklike Nederlandse Akademie het die vergadering toegespreek oor 'Die ontwikkelingsfisiologie van die oog'. 'n Groot en belangstellende gehoor het hierdie interessante lesing bygewoon. Ongelukkig kon prof. H. W. Snyman, Hoof van die Departement Interne Geneeskunde van die Universiteit van Pretoria en voorsitter van die Afdeling Geneeskunde van die Akademie, weens onvermydelike omstandighede nie daardie vergadering bywoon nie. Professor Snyman sou die vergadering toegespreek het oor ‚Geneeskundige onderrig in Afrika'—'n onderwerp waarop sy onlangse besoek aan verskeie mediese skole en inrigtings in Afrika interessante lig sou kon werp.

BOOK REVIEWS : BOEKBESPREKINGS

LEUKAEMIA

Etiology and Treatment of Leukemia. Proceedings of the First Louisiana Cancer Conference. Edited by Walter J. Burdette, Ph.D., M.D., F.A.C.S. Pp. 167. 14 figures. South African price 34s. St. Louis: The C. V. Mosby Company. 1958.

This book contains some of the proceedings of the First Louisiana Cancer Conference. The exact date of the Conference is not given. There are half a dozen papers by active research workers on the aetiology and diagnosis of leukaemia and as many on the treatment of the disease.

Of chief interest in the concept of the aetiology is the work of Gross and Woolley dealing with the possible place of viruses in the production of leukaemia. There seems little doubt that certain varieties of animal leukaemia may be so caused, but it is still a long way from mice to men. As regards therapy, there is little new despite the great efforts at present under way. The drugs used are discussed and the indications for them enumerated. The contribution by Till from the Chester Beatty Research Institute on their work on the use of chlorambucil and bisulphan demonstrates Anglo-American cooperation in a pleasing way. The record too of the discussions which followed the papers adds greatly to the value of this book. There is surprisingly very little on the possibility of transplantation of marrow after the destruction of the malignant process by radiation or other means. Nor is there as yet any sign of a major 'break through' in the efforts to cure this disease. The best that can at present be said is that some amelioration is possible, the average duration of survival has been increased, and some of the patients are more comfortable.

The book is nicely produced and reasonably priced. It is a pity that the bibliography is not presented in alphabetical order; it would have provided a useful summary of current medical research in this field. This volume succeeds nevertheless in being an 'arrested instant' in the progress to what we all sincerely desire—a cure for this dreadful disease.

C.M.

MODERN TRENDS IN PAEDIATRICS

Modern Trends in Paediatrics. 2nd series. Edited by A. Holzel, M.D., D.C.H. and J. P. M. Tizard, M.A., B.M., M.R.C.P., D.C.H. Pp. xxiii+372+(13). 46 figures. 78s. 9d. + 1s. 9d. postage. London: Butterworth & Co. (Publishers) Ltd. South African office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

This is the second in the series of *Modern Trends in Paediatrics* and is intended as a companion volume to the first, published in 1951. There are 23 contributors, the majority from various British medical schools, although there is a distinguished minority of authors from the USA and Europe as far afield as Moscow. This well-groomed publication belongs neither to the text-book nor the 'Recent Advances' or 'Year Book' categories, but comprises a series of discussions on selected paediatric problems or on topics which the authors (not all of whom are paediatricians) consider to have a direct bearing on modern paediatric practice. In the first place each subject is dealt with factually in the light of recent research and a comprehensive bibliography is included at the end of every chapter. Thereafter the author expresses his own views on future trends and developments which would be most likely, in his opinion, to shed further light on the particular subject discussed. This forecast of the future is a particularly interesting aspect of the work, since it must stimulate thought in the reader—thought which may readily be translated into action.

This is a book which is, perhaps, primarily for the specialist, but the general practitioner with a leaning towards paediatrics will find much to interest him.

H.L.W.

PRINCIPLES OF RESEARCH

Principles of Research in Biology and Medicine. By Dwight J. Ingle, B.S., M.S., Ph.D. Pp. ix+123. 30s. net. London: Pitman Medical Publishing Co. Ltd. 1958.

One is prejudiced from the start in favour of a book on the philosophy of science which quotes freely from such a practical philosopher as Lewis Carroll. Although Professor Ingle lacks some of the clarity of expression of his distinguished predecessor the contents of his book make it well worth reading. It is appropriate that it should be published at a time when many scientists, unsatisfied with the mere cataloguing of facts, are speculating on the nature and purpose of science itself.

Professor Ingle sets out to provide general concepts of the aims, methods, and limitations of science, with particular reference to biological research and to the uses and limitations of statistics. He explores many common fallacies, which the scientist cannot afford to ignore, and he gives detailed advice to scientists of all degrees on the organization and conduct of their work. The logic of the text is sound although many of the postulates are, probably intentionally, controversial. Much concentrated argument is condensed into small space and the reader may find it necessary to read the book more than once in order to derive the maximum benefit from it.

Probably the greatest value of this book is that it expresses in words a number of general ideas fundamental to science, which

individual scientists may never have attempted to define. The book can be read with profit, though not with ease, by anyone engaged in biological research.

A.W.S.

DERMATOLOGY

Dermatologie und Venerologie—einschliesslich Berufskrankheiten, dermatologischer Kosmetik und Andrologie. In 5 Bänden. Herausgegeben von Prof. Dr. Dr. h.c. H. A. Gottron und Prof. Dr. Dr. h.c. W. Schönfeld. *Band II. Teil 1. Physikalische Behandlung—Dermatologische Kosmetik—Krankheiten noch unbekannter Herkunft nach ihrer Morphologie I*—Bearbeitet von zahlreichen namhaften Fachgelehrten. xvi+388 Seiten. 388 teils farbige Abbildungen. Ganzleinen DM 173.00. Subskriptionspreis DM 138.40. *Band II. Teil 2. Krankheiten noch unbekannter Herkunft nach Ihrer Morphologie II—Krankheiten mit bekannten Erregern.* xvi+670 Seiten. 277 Abbildungen. Ganzleinen DM 154.00. Subskriptionspreis DM 123.20. Der Subskriptionspreis für das Gesamtwerk gilt bis zum Erscheinen des letzten Bandes. Jeder Band ist einzeln zum Ladenpreis käuflich. Stuttgart: Georg Thieme Verlag. 1958.

In the old days the chief reference work in German in the field of dermatology was the mammoth *Handbuch der Haut- und Geschlechtskrankheiten* edited by Professor Jadassohn. The firm of Georg Thieme of Stuttgart has taken on the task of carrying on this tradition of vast anthologies, and this is the first published volume, Volume 2, of the final 5 volumes planned. It consists of 2 parts, each a hefty text-book of several hundred pages.

Part 1 deals with the treatment of skin diseases with physical modalities—X-rays, radio-active substances, heat, ultraviolet light, ultrasonic vibration and climatic therapy. Each special therapeutic aid is considered by a specialist in the field. On the whole a conservatively cautious attitude is adopted. The articles are illustrated by graphs and mathematical formulae. The dangers inherent in any form of ionizing irradiation are adequately stressed.

Part 1 also deals extensively with dermatologic cosmetics and with skin sensitization caused by many of them. The formulae of the most treasured perfumes, body-deodorants, depilatories and other beauty aids are laid bare ('Chypre Orientale' consists of 25 ingredients). Finally, in part 1, there are several excellent chapters on numerous skin diseases of still undetermined aetiology—psoriasis, lupus erythematosus, the erythrodermias, dermatomyositis, the pemphigus group, and many more.

Part 2 continues with diseases of unknown origin—lichen ruber, planus and nitidus, the haemorrhagic diatheses, and scleroderma and its variations—and then a very full and well-written section on skin disease of known aetiology—those due to the common parasites, the mycotic diseases, and the inflammatory conditions caused by micro-organisms and viruses. There is also a chapter on sarcoidosis, in which the various theories of the underlying causation are presented with laudable objectivity.

Thieme's familiar light-blue dust-jacket conceals a very handsome binding and the printing, paper and illustrations are of a high standard. There are voluminous references.

The complete work will surely embody a vast accumulation of dermatologic knowledge. Its happy possessor may well approach his patients with a confident air only, perhaps, to feel somewhat helpless when confronted by a case of chronic eczema!

C.K.O'M.

CORRESPONDENCE : BRIEWERUBRIEK

CEREBRAL PALSY

To the Editor: According to a recent radio announcement, the United Cerebral Palsy Association (U.C.P.A.) has claimed that very little is being done in South Africa for victims of cerebral palsy as regards adequate medical treatment. I understand that this Association is about to conduct a 30-day fund-raising campaign throughout the country in order to raise funds to develop the necessary medical facilities.

The public has the right to know the facts of the situation before supporting such a campaign. Cerebral palsy rehabilitation work in this country, as in every country in the world, is of recent origin. The first treatment centre for the cerebral palsied in the Union was opened only 11 years ago. Since then, however, great strides have been made. Today, under the auspices of the National Council for the Care of Cripples which, with its affiliated bodies administers all cerebral palsy centres throughout the country (except the U.C.P.A.'s clinic at Townsview), there are 7 centres for the care, education, and treatment of the cerebral palsied. These centres, situated in all the Provinces, are assisting over 700 cases, and offer treatment which compares favourably with that in Britain, America, and on the Continent. An eighth centre is being opened in Durban during the next few weeks.

Three leading experts in Britain in the cerebral palsy field have visited South Africa during the last few years, and given us invaluable guidance—Mr. G. Pollack of Edinburgh, and Dr. and Mrs. K. Bobath, of London. All have been deeply impressed by what is being done here. I myself have visited Britain on several occasions during the last 6 years to study the latest developments in treatment. I can say quite categorically that we have nothing to be ashamed of in our modes of treatment in this country. It is a complete misstatement to say that little adequate medical treatment is being carried out in South Africa.

The National Council for the Care of Cripples has no quarrel with the U.C.P.A. as far as the work of its clinic on behalf of the ineducable is concerned and is anxious to offer its support to this work. Those of us who understand the nature of cerebral palsy are sometimes alarmed at the optimistic prognoses suggested in some of the Association's propaganda, and at such sweeping and incorrect generalizations as referred to above. We feel that the public should be told plainly that the National body correlating all cerebral palsy work in this country is the National Council for the Care of Cripples in South Africa, working through its Cerebral Palsy Division. The U.C.P.A. is the only body working in this field that is *not* affiliated to the National body. Had it been affiliated, much of this apparent working at cross purposes could have been avoided. Funds collected by the U.C.P.A. are administered by this organization, and do not go to support the major cerebral palsy institutions in South Africa.

The National Council for the Care of Cripples is a highly responsible body which has full Government support for its cerebral palsy, and other work. It has not officially and publicly joined issue with the United Cerebral Palsy Association because it does not wish to give the impression that it is working against any group which is seeking to assist any cerebral palsied person. At the same time it is evident that by its misleading propaganda the U.C.P.A. is causing a good deal of confusion and uncertainty in the public mind and, in the long run, is defeating its own end and that of the Council, both of which wish to serve the interests of the cerebral palsied.

As someone who has been working for some years with this kind of handicap, I appeal to this body to work with, and not against, all other bodies in South Africa who are serving the same cause. I want to make it quite clear that the U.C.P.A. does not speak for the majority of the cerebral palsy associations which are doing such splendid work in this country. It speaks for itself alone—an isolated and independent organization which up to now, for reasons best known to itself, has decided to 'go it alone'.

104 van Riebeek Medical Building
Schoeman Street
Pretoria
31 July 1959

Ben Epstein,
Chairman,
Board of Management,
Pretoria School for Cerebral Palsy.

DIRECT ARTERIAL SURGERY

To the Editor: It was with great interest that I read the article by Prof. J. H. Louw and Mr. L. Blumberg[1] on this subject. In the main this article reflects the procedures now adopted in London by Professor Rob. I should, however, like to comment on some aspects of this paper.

Professor Louw and Mr. Blumberg state that in abdominal aneurysms aortography, though not essential, may be of great value. Professor Rob maintains that this is rarely necessary since not one of the abdominal aneurysms in his series had involved the renal vessels. Professor Rob further states that if the fundus of the aneurysm can be felt below the zyphisternum, the aneurysm does not involve the renal vessels.

The fact is worth stressing that wound disruption is a very real problem and occurred 3 times in 13 cases at Groote Schuur Hospital even though the greatest care had been taken in closing the abdomen.

The conservative approach to peripheral arterial grafting is rightly stressed by Professor Louw and Mr. Blumberg. This conservative attitude is however not universal, e.g. De Bakey.

Although De Bakey claims that results with prostheses are as good as homografts for peripheral vessels, no one has yet produced a worth-while series of cases to show patency of the graft at the end of one year. Certainly Professor Rob has not been able to reproduce De Bakey's results.

With regard to thrombendarterectomy below the inguinal ligament we should reserve judgment on Cockett and Norman's views.

At present it would appear that using a homograft is preferable to thrombendarterectomy for peripheral vessels.

Martin is now using deproteinized bullock's carotids inside a plastic prosthesis in animal experiments. He is hoping that this will be applicable in humans.

Mannie Stein

306 Colonial Mutual Buildings
West Street
Durban
28 July 1959

1. Louw, J. H. and Blumberg, L. (1959): S. Afr. Med. J., 33, 576.

ROAD ACCIDENTS AND THE HARD OF HEARING

To the Editor: The article by Mr. J. G. du Toit[1] on the important subject of road accidents which was published in the *Journal* of 4 April 1959, is of great value, and it is one of the first attempts to awaken the profession to the seriousness of this problem.

The main substance of the article is a very fair assessment of the problem, and there are many sensible suggestions for grappling with this disastrous state of affairs. However, I must disagree with the sweeping statement contained in his paragraph on the issuing of driving licences to those persons who suffer a disability; I refer particularly to those who are unfortunate enough to be deaf or hard of hearing.

At a recent meeting of the Technical Sub-committee of the National Council for the Deaf, Mr. du Toit's article was considered from the point of view of whether deafness as such was a reasonable cause for the refusal of a driver's licence.

The National Council for the Deaf has had this subject under consideration since 1950, and it holds the view that deafness in no way is a disability which would increase a driver's liability to accidents since statistics tend to show the reverse.

The following extracts from official correspondence support this view:

From the Director, National Road Safety Organization of South Africa (26 *August* 1950)

'We have no objection to use being made of the opinion of this Organization that there should be no discrimination against drivers with impaired hearing if the application is for a licence to drive a private motor vehicle—but an advanced standard of hearing should be required for drivers licensed to drive public transport vehicles.'

From The Accident Insurance Council of South Africa (3 *November* 1950)

'My Council has no regulations prohibiting the granting of insurance to deaf drivers and for this reason there is no other alternative but for each company to treat the matter as a domestic one . . . in many instances companies do not load the premiums in respect of deaf drivers.'

From the Secretary for Transport, Union of South Africa (27 *August* 1953)

'It has been decided not to write in the new Ordinance a specific exemption in respect of applicants who are deaf and who wish to obtain a driver's licence. It has been found that throughout 16 years of testing applicants for driver's licences, not one case has been refused a driver's licence solely on this ground, and in view of the provision made for appeals being heard where such applicant is refused a licence for which he has applied, it is considered that adequate safeguards exist against any possible penalizing of the hard-of-hearing drivers. This attitude is, I understand, also favoured by the other Provinces. This Department agrees with the views of the Provinces.'

From the Provincial Secretary, Transvaal (22 *February* 1954)

'This Administration's policy is that deafness by itself is not sufficient reason to refuse a driver's licence . . . a person who has been refused . . . has the right of appeal to the Administrator.'

From the Secretary, National Institute for the Deaf, London (11 *February* 1954)

'I have no knowledge of any case where deaf drivers have applied and have been refused licences just because they were deaf. There have been cases where they have been refused because their standard of driving was not good enough anyway. In the opinion of the Institute and from our experience deaf drivers are as safe—if not safer—than many of the hearing drivers we have in this country and we have never known of a case in which a deaf man was involved in an accident where *deafness* was the cause of the accident.'

From the Organizing Secretary's Administrative Report to Executive Committee of the National Council for the Deaf (*August* 1955)

'The Cape Provincial Council has approved the new uniform Vehicle Ordinance—which included the provision suggested by the Council—that deafness of itself should not preclude any person from obtaining a driver's licence.'

Mr. du Toit's article received widespread publicity in the lay press. It would be most unfortunate if the deaf and hard of hearing should be prejudiced by the erroneous statements in this otherwise sound article.

P. Meyrick

112-114 County Building
Church Street
Pietermaritzburg
30 July 1959

1. du Toit, J. G. (1959): S. Afr. Med. J., 33, 296.

COPY OF LETTER DATED 28 JULY 1959, TO MR. E. R. GRIFFITHS, CHIEF INSPECTOR OF SCHOOLS, TRANSVAAL, FROM DR. BEN EPSTEIN, CHAIRMAN, SOUTH AFRICAN ORTHOPAEDIC ASSOCIATION (M.A.S.A.)

Further to our discussion of 21 July 1959, I am now writing to you formally to ask you to investigate the problem of 'home teaching' for Transvaal children.

It is within my experience that a certain number of children suffering from sub-acute illnesses are deprived of schooling for considerable periods of their lives. I have known children to be away from school for periods of 3 months, 6 months, or even a year; during this period, if they were not patients in a hospital where schooling is provided, they were completely deprived of education.

Illnesses which may keep a child away from school occur not infrequently, e.g. nephritis in its various forms, rheumatic fever with involvement of the heart, primary tuberculosis (non-infectious), poliomyelitis, accidents, and various other conditions too numerous to mention. A child may be unable to go to school for a long time after the period required for active hospital treatment.

I am certain that many difficulties which these children have subsequently in their schooling can be traced back to this period of absence from schooling, when they have missed fundamental educational training.

As you know, the idea of home teaching is not new. There are many countries in the world that provide for children to carry on with their education while they are not completely well. I do not think that the cost of this service should be a primary consideration. It would be well compensated by the economic asset of eliminating waste due to lack of education, and the development of bad habits resulting in loss of interest in study.

I had intended writing to you in my private capacity, but in view of the importance of the subject I write on behalf of the Paediatric Association.